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THE PATHOLOGY AND THERAPY OF THE TONSILS IN THE LIGHT OF THEIR PHYSIOLOGICAL FUNCTION.

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As long as we were in the dark concerning the physiology of the tonsils we had no foundation upon which to build the pathology of these organs. To formulate correct conclusions regarding their therapy was difficult, because most of the data with which to do so was empirical and often contradictory. This was confusing. Until recent times the therapy of the tonsils has been a very much disputed field.

Indication for operation was evident enough when the diseased and enlarged tonsils gave rise to deleterious effects. One was inclined to hold the diseased tonsils responsible for a whole series of infections, but in these cases it was unnecessary to accord the physiological function of the tonsil any further consideration because it was presumptive that such function was already lost. Hence the removal of the tonsils could be advised.

There was great danger, however, that our lack of information as to the physiological function of the tonsils might prove of sinister significance. For if, on the one hand, the usefulness of these organs could be denied, and on the other hand, their deleteriousness affirmed, it was but a short step to their summary condemnation. That this danger was not purely imaginary is shown by the reports from America where the enucleation of the tonsils has gone on to such an extent that it is spoken of as "the slaugh-

*Translated by Dr. Philip Frank.

ter of the tonsils." The fate formerly accorded to the turbinates was now meted out to the tonsils.

My investigations on the path of the inspiration air current through the pharnyx (*Arch. für Laryngol.* Bd. 30, Heft 2) have afforded a basis for the view that the tonsils may serve a valuable function in breathing. It would seem necessary, therefore, to review the pathology and therapy of the tonsils from the standpoint of their physiology.

Let us take the view that the tonsils are transudation glands whose function it is to supply water to the relatively dry inspiration current of air going to the lungs. It is clear that in cases where the general condition is related to a change in the properties of the lymph, those organs that are concerned with the flow of the lymph may easily become diseased. That is to say, since the old humoral pathology has been rejuvenated under the name of serology we have to bring the following points to our attention: that in the infectious diseases which here come under consideration, the lymph is the main breeding place of various organisms. One can easily conceive that this lymph, the nutritive quality of which becomes vitiated through the action of micro-organisms, may produce disease in the tonsils during its passage through them. Even if the tonsils withstand the disease-producing influences, they may still show evidence of the pathological properties of such lymph. One of these properties is heightened coagulability and this will often manifest itself on the surface of the tonsils. It is evident that this must occur when the lymph makes its exit from the tonsils and coagulates on contact with the air. Coagulated material will then be present on the surface and in the excretory ducts of the tonsils.

To be sure, normal lymph also coagulates on exposure to air. But it requires some time to do so. Under ordinary circumstances the tonsillar fluid will long have disappeared before such coagulation would even begin to take place. Even if the lymph were to coagulate immediately upon its excretion from the tonsils other special factors are necessary to cause a retention of the coagulated material at the points of excretion. For instance, the thin stratum of adenoid tissue which we find in the mucous membrane of the pharnyx and of the nose and trachea, also allows lymph to pass through it. But this "diaphoretic fluid", as we might well call it, makes its appearance on a smooth surface where its retention is very difficult. Only under special conditions as, for example, in severe forms of diphtheria can the coagulated material be seen in the form of a pseudo-membrane. Otherwise, as a rule, the

masses of fibrin are found on the tonsils where they cling to the longer, more coiled, excretory ducts.

Not alone infectious diseases cause a heightened coagulability of the lymph. They are the most important, but not the sole, factors which may produce this pathological change. It is further possible that such lymph may possess pathological color which may show itself in the coagulated material on the tonsils.

This short review suffices to indicate the behavior of these glands as organs of excretion. This applies to all tonsils but as the faucial tonsils are open to direct inspection and hence have been accorded most attention, we will deal with them in particular.

It is known that the form of the tonsils depends upon a thickening of the mucous membrane as a result of enclosure of round cells and the formation of lymph nodules. During the round cell infiltration, the tissue of the tunica propria changes into a reticulum resembling in every way the stroma of lymph glands (Disse). Inasmuch as adenoid tissue cannot be differentiated from lymphoid tissue we may say that the histological structure of the tonsils is the same as that of lymph glands but the former do not pour their circulating lymph, as do the latter, into a lymph vessel, but into the pharnyx. Hence, we find in the tonsils, instead of an efferent vessel, a number of ducts through which the lymph flows into the pharnyx. Thus the tonsil may be regarded as an exposed or modified lymph gland.

This at once brings up the question of the afferent lymph vessels to the tonsils. According to Disse the lymph vessels of the soft palate, like the veins, form two plexuses: The dorsal plexus is connected with the lymph vessels of the floor of the nasal cavity; the anterior plexus with the lymph vessels of the tongue. The plexuses empty themselves through little vessels lying lateral to the tonsils and coursing towards the lymph glands of the neck which lie to the side of the pharnyx on the carotid (Heimann's Handbuch, Bd. 2, p. 43). According to Disse these little branches are taken up by the lymph vessels of the tonsils. Certain experiments, however, point towards a different relationship between the tonsils and the lymph vessels.

Henke (*Deutsche mediz. Wochenschrift*, No. 33, 1913) found that after the injection of a suspension of soot under the mucous membrane of the floor of the nose he was able, upon extirpation of the tonsils 24 to 36 hours after the injection, to find particles of the soot within the tonsils. Similarly, he was able to demonstrate the soot that he had injected under the buccal mucous membrane, in both tonsils and also in the lingual tonsil after a certain

time. If too long a time elapsed the soot particles could not be demonstrated in the tonsil tissue; hence, the supposition that they had been excreted.

While other investigators have not always been able to substantiate these results, it is only the positive findings that are operative. The negative do not prove the contrary.

Let us understand what happens to the injected soot. First, the soot particles must find their way to the dorsal plexus of the velum. Then they must enter the branches of the vessels running laterally to the tonsils towards the lymph glands of the neck. Some of these particles, however, do not reach these glands but by a collateral path reach the tonsil directly. This means that the collateral path is free and that, contrary to general opinion, the lymph current in the lymph vessels is directed to the tonsils. Furthermore, experiment has shown that the injected soot particles are not retained in this collateral route, which must be conceived in the form of short, rather broad lymph channels penetrating the capsule and emptying into the hilus of the tonsil. It is hard to imagine that the disappearance of the soot particles, as found by Henke, takes place in a retrograde manner. The supposition is, rather, that these particles have passed through the adenoid tissue and have been carried off through the lacunae into the pharnyx. This agrees with the places where Henke found the particles in the microscopic sections.

It should be observed that this investigator did not find the soot particles within the leucocytes. He therefore assumed that they had been carried along with the lymph current. His findings agree fully with our own assumption that the function of the tonsils is to allow the passage through them of lymph in order to give up its water to the inspiration air current.

The adenoid tissue that we find outside of the tonsils in the air passages is histologically identical with tonsil tissue. But it is broadened out into a thin layer and does not receive its lymph supply from larger vessels, as do the faucial and probably also the other tonsils. It is probable, therefore, that this adenoid tissue receives its fluid from lymph spaces in indirect relation to the blood vessels. Therefore, in connection with this thin layer of tissue, the lymph "sweats through", as we might say, while in connection with the tonsils we must understand the existence of a lymph current.

In both instances, however, the propelling force is the pressure to which the lymph is subjected. In the last analysis this pressure is concerned with the blood pressure and it may be greater

in the lymph vessels than in the lymph spaces. At any rate, the moisture of the pharyngeal mucous membrane must be regarded as being under the indirect influence of the blood pressure.

From this point of view a diminution in the moisture of the pharnyx and especially of the air passages is to be looked upon as a natural sequence of a decrease in blood pressure. When this decrease has only reached a certain point, it will manifest itself as a feeling of dryness, ordinarily known as thirst. The importance of the localization of the sensation of thirst in the pharnyx is that it shows what an important role the tonsils play in the moistening of the upper air passages. I know that the proposition is not as simple as I have here presented it and that there are still other questions regarding the problem of thirst that await solution. But it is worthy of observation that in Mayer's work "Essai sur le Soif", Paris, 1901, where the problem is considered exhaustively, such an explanation is not appreciated.

Just as there may be a decrease, so may there be an increase in the amount of lymph in the tonsils, under the influence of the blood pressure. That does not mean that the amount of lymph in the pharnyx need also be increased. If the excretory ducts of the tonsils remain patent, a heightened pressure can only manifest itself as a result of a more rapid circulation. This rapidity, which is proportional to the square root of the propelling force, is, however, determined by the resistance to be overcome. In a tissue such as the tonsil this resistance, under varying conditions, is difficult to measure. Probably the increase in blood presssure causes a greater flow of lymph through the tonsils. Perhaps there may develop a lack of balance between the inflow to and the outflow from the tonsils. In such an event, if the outflow is relatively less than the inflow, lymph stasis must result. The elasticity of the tonsils as a whole will then determine the degree of their enlargement.

Slight enlargement of the tonsils, of short duration, may be regarded as within normal variation. Greater, more persistent enlargement may be regarded as incapable of retrogression. In this way, enlargement of otherwise normally functionating tonsils may occur. The question arises, can the so-called hyperplasia of the tonsils so often found in children be explained in this manner? The so-called "lymphatic constitution", so frequently invoked as an explanation, may thus be abandoned and an attempt made to elucidate these deviations upon the basis of blood pressure variations.

Where the tonsils are enlarged but one is convinced that the tissue is not frankly diseased, in the general acceptance of the term, the question of operation should be confined at most to a reduction in the size of the tonsils, and then only when such procedure is thought necessary for proper breathing or speech. This should be the general rule, for it has been shown that these hyperplasias tend to spontaneous disappearance at the second dentition.

Not only the quantity, but also the quality, of the lymph may be of importance in tonsillar enlargement. The hypertrophy of the tonsils occurring in leukemia and pseudoleukemia (Schrötter) is well known. That in these cases it is the lymph which is to be held responsible is evident from the fact that there is similar hypertrophy of all those organs in which lymphoid tissue predominates—the spleen, lymph glands, bone marrow, etc. Furthermore, it becomes clear that in pseudoleukemia the number of leucocytes, upon which considerable stress has been laid, is of absolutely no importance in the production of such hypertrophy. We must assume that it is the chemical properties of the leukemic and pseudoleukemic lymph that are of etiology in the hypertrophy of the tonsillar tissue. Of course, at first this consists of a hyperplasia of the cellular elements; we would be inclined to term the irritation nutritive in character. But the involvement of the interstitial connective tissue with resulting induration, shows that this hyperplasia is not merely a functional affair, but that the leukemic and pseudoleukemic hyperplasia is decidedly pathological.

In the light of the physiological function of the tonsils it is comprehensible enough that upon the circulation through the tonsils of a considerable amount of pathological lymph, they should react by hypertrophy. The same may be said with just as much justice in regard to all other chronic blood diseases in which the lymph possesses irritative properties in relation to adenoid tissue.

The subject becomes even plainer when we recall the chemical substances that give to the lymph these irritative properties. Iodin is the most important of such substances. Iodin when given in small doses internally may produce an acute enlargement of the tonsils. Investigation may reveal many other such substances in the lymph.

It would seem that adenoid tissue belongs to those structures which most easily undergo inflammation. An inflammation of the tonsil tissue—a tonsillitis in the strict sense of the word—is only conceivable when the causative factor is derived not from within, but from without, the tonsils. Foreign bodies, solid as well as li-

quid, or conditions of gas formation, may injure the tonsil tissue and initiate an inflammation. But that, however, is independent of the function of the tonsils. The disturbance in their function is secondary. Thus, as a result of the inflammation, the excretory ducts may become occluded and give rise to lymph stasis. I do not mean to infer that every tonsillitis, the causation of which lies within, must necessarily originate from the blood, i. e., by the hematogenous route. It suffices that only the lymph circulating through the tonsils possess the irritative, inflammatory properties. This is also conceivable when the causative inflammatory factor is situated peripheral to the tonsils and gives the lymph its deleterious properties, for as the tonsils derive their lymph from the lymph glands (centripetally) an inflammation of any such lymph gland may be confined to the lymphatic area entirely. Under these conditions we may speak of a tonsillitis of lymphogenous but not of hematogenous, origin.

This explanation applies very well to those tonsil inflammations that are not infrequently observed after intranasal operations. Just as in Henke's experiments the particles of soot found their way to the tonsils, so may inflammatory substances find their way to the tonsils. It should be observed that angina dentaris (Heymann's Handbuch, Bd. 2, p. 550) following operations in the mouth is thus analogous to inflammations following intranasal operations.

We have been inclined to think that tonsillitis following intranasal operations must necessarily be ascribed to infection. Yet we often see such inflammation in cases where the strictest precautions against infection have been taken, even in cases where the employment of the galvano-cautery has left no open wound. Although, as a general rule, there is no inflammation without microbic invasion, there may occur non-bacterial changes in the lymph which may produce inflammation of adenoid tissue, as in iodin intoxication. Lymph gland inflammation has also been observed after operations under the strictest asepsis in other parts of the body, as well as after contusions without any external wound. Why may not this be the case with the tonsils, which may certainly be considered regional lymph glands?

The lymph may possess a certain virulence aside from that produced by micro-organisms. Perhaps excessive temperature is conducive to such a change in the lymph. This would explain the occurrence of tonsillitis after galvano-cautery operations on the lower turbinate. But even under physiological conditions the

lymph may undergo a certain virulence—take, for instance, the tonsillitis sometimes seen during menstruation (tonsillitis katamenialis and menorrhagica). The occurrence of tonsilitis in various blood diseases may be due to a temporary virulence of the lymph, as in leukemia and other diseases of the blood.

However, in the great majority of cases, the inflammatory property of the lymph must be ascribed to infection. The question as to whether it be the bacteria themselves or their toxins that are carried to the tonsils need not here be discussed. It is not of much importance. Of greater importance is the question as to how the infectious material invades the tonsil through the lymph, that is to say, from within. It is also possible that the tonsil may be invaded from without, that is to say, from the pharyngeal cavity where bacteria are always present in sufficient quantity and variety.

The question, then, why a tonsillar infection is not always of pharyngeal origin may be answered in various ways. We are brought back to the question of the extravasation of leucocytes from the tonsils which has been known since the work of Stöhr in 1884. Metschnikoff's teachings on phagocytosis were first utilized for an explanation of this question. The idea was attractive enough. It was very simple to assume that the leucocytes ingested the bacteria and then destroyed them. Bloch (Heymann's Handbuch, Bd. 2, p. 554) even speaks of the mobilization of countless numbers of leucocytes, like a defensive army, which upon the bacterial attack, make their appearance on the scene to destroy the attacking host. But the question arises: Of what use can the phagocytosis be since the leucocytes at once pass from the tonsils into the pharnyx? It does not matter whether these cells first ingest the bacteria or whether they travel along mechanically, as B. Fränkel pictures it, for the extravasated leucocytes do not encounter conditions most favorable for their own existence or for resisting their own destruction.

Discarding this view of the role of the leucocytes we might, as suggested by Rosbach, seek their power of protection in chemical substances excreted by them. But here it is hard to understand why the wandering cells in the lymph and blood do not also eliminate such substances. If they do, then the lymph entering the tonsils would already possess a protective power and for the leucocytes in the tonsils there would be nothing left to do. Aside from the fact that the lymph, on the contrary, is an excellent nutritive medium for the multiplication of various germs a further question arises as to what use such bactericidal power the ton-

sillar fluid would have, inasmuch as this fluid passes out in a constant stream? If the circulation of the stream were retarded it would suffice to allow invasion of the tonsillar tissue with organisms; but as it is, any infected material that happens to be carried in at one moment is swept out the next moment.

The trouble with the above explanation is that we have retained our conception of the leucocytes as Stöhr saw them through the microscope. A little reflection must teach us that we must not conceive of these cells as wandering along on a dry path. We can only think of them as swimming along, so that incidentally Stöhr's discovery included also the extravasating lymph stream which we accept in connection with the inspiration air current through the pharnyx. This lymph stream commands attention because of its water content. As to the cells that are extravasated with it, they die shortly after extravasation. No significance need be attached to them whatever. Therefore, we regard the lymph stream, independently of its wandering cells, as a sufficient protection against infection from the pharnyx.

It is evident that this proposition is correct only for adenoid tissue, where the lymph stream may be regarded as being under a certain pressure. As soon as the lymph exudes and collects in the lacunae and spaces of the tonsils, the conditions change. Of course, in these excretory passages the fluid is being constantly renewed, but in a slow and irregular manner. A transient infection of the lymph in these passages is possible. This agrees with most of the results obtained in the investigations of the lacunar secretion. Meyer states that he found micro-organisms in the lacunar secretion from the depth of healthy tonsils but in much smaller amount than in the superficial secretion. Among these organisms he was able to isolate a streptococcus greatly resembling the streptococcus pyogenes but which was not pathogenic; some cocci arranged as diplococci but which he was unable to identify as pneumococci; staphylococcus pyogenes aureus and albus; some fat, thick cocci and the streptothrix. If we agree with Meyer that the staphylococcus is a rather innocent accompaniment in infections, no pathogenic bacteria were present. Even the streptococcus pyogenes which Meyer regularly found in the tonsil secretion and which he holds as the cause of angina, cannot be proved to have come from the pharnyx. For example, it is quite possible for this organism to have reached the tonsil through the lymph stream from the lungs.

But suppose we assume that the pathogenic bacteria invade the

tonsil from the pharnyx. We would expect, first, a tonsillitis followed by inflammation, through the hematogenous route, of the remaining adenoid tissue. Experience teaches, however, that the follicles on the posterior pharyngeal wall—the islands of adenoid tissue—which can only become inflamed by way of the blood stream, are inflamed simultaneously. Hence, we must assume against a retrograde infection, from the pharnyx in a direction opposite to the flow of the lymph stream; at least, while such a theoretical possibility cannot directly be excluded, it seems, however, very improbable.

It would appear that thus far, in the pathology of the tonsils, too little weight has been placed on this very point: the improbability of a retrograde infection of the tonsils. Not enough attention has been given to the lymph stream which passes through the tonsils.

To say that the leucocytes passing out of the tonsils possess a protective function against infection from the pharnyx is a fantastic notion. Some authors even advance the view, for example Bouchard and Lermoyez, that cold acts like a traumatic shock and produces a stasis of the leucocyte stream with resulting exposure of the lacunae of the tonsils to microbic invasion. The whole thing seems much simpler if we assume that the tonsillitis is secondary to infection through the nasal mucous membrane, although several investigators have claimed that the nasal secretion possesses certain bactericidal properties.

The truth must be confessed, however, that we do not know beyond question how, in tonsillitis, the infection occurs. To hold the tonsils under suspicion is not entirely justified because their guilt has by no means been proven. On the other hand, the improbability of an infection in an opposite direction to the lymph current would point to the culpability of the tonsils. Then again, from the corresponding inflammation of all the mucous membranes containing adenoid tissue it is hard to find any other explanation than the hematogenous route of infection.

That in tonsillitis we are dealing with an infection will not be denied. The clinical course of the disease all substantiates this. The sudden onset, following an incubation period without any special symptoms on the part of the tonsils, often accompanied by a chill, has a typically infectious character. So also the fever, often falling by crisis, and the general symptoms point to infection.

But what kind of infection? Bacteriology should be able to solve this question but it does not, for its findings are by no means in agreement. Edmund Meyer reached the conclusion, on the basis of 56 cases examined by him, that the majority of cases of

tonsillitis were caused by the streptococcus pyogenes; that the staphylococcus was only rarely the cause of the disease. The diphtheria bacillus as well as the pneumococcus may produce a clinical picture of tonsillitis. The findings have, therefore, not been conclusive and later investigations, as far as I know, have brought no change in the situation. Apparently, the same streptococci are encountered in various infections, differing so from one another, that these organisms cannot be held to be identical. The proposal has, therefore, been made that these infections be grouped under the general heading of streptomycoses. It may be said that from the bacteriological findings, tonsillitis is not characterized by any definite bacteriology, and on this basis it need not be regarded as a disease *sui generis*.

Clinically, we find that the period of incubation varies greatly. Bianchi states that it may vary from a few hours to three days; Descorgu gives it as being from half a day to thirteen days; Wolberg, from three to five days. Variations are seen also as regards the height of the fever, the severity of the general symptoms and their duration. But differences are especially notable as regards the so-called complications of tonsillitis: general pyemia and sepsis, pericarditis, myocarditis, endocarditis, ulcerosa and verrucosa, thrombosis and phlebitis, erysipelatous inflammation of the neck, serous and purulent pleuritis, pneumonia, infarcations of the liver and spleen, infiltration of Peyer's patches, perityphlitis, nephritis, orchitis, ovaritis, meningitis, erythema etc.

How can we believe that the same disease may most often disappear without traces in a few days and also give rise to such widespread complications? Would it not be simpler to assume that we are dealing with a number of infectious diseases which have the tonsillitis as a symptom in common?

When a lymph gland is inflamed we do not regard that as a disease *sui generis* because we know that the adenitis may be a consequence of various infections. It may be hard to look upon inflammation of the tonsils from this point of view. Even Bloch, although he admits that many diseases predispose to tonsillitis, hesitates to accept the view of Landouzy that in the majority of cases the tonsillitis is only a symptom of systematic infection. Nevertheless, no other theory so happily explains the diversity of the bacteriological and clinical findings.

In the light of the physiological function of the tonsils this theory is perfectly logical; for, if the lymph excreted through the tonsils contain inflammatory and irritating substances, it is suffi-

cient to give rise to a tonsillitis. It does not necessarily follow that in every infectious disease the lymph possesses such properties and must produce a tonsillitis. But it is very probable that in many such infections the tonsils become involved. Just as easily may the adenoid tissue in the mucous membrane in the neighborhood of the tonsils show inflammatory swelling as a reaction to infection. Farlow advises that in any tonsillitis the cause should be sought in the immediate neighborhood. But rarely will it be found there. The cause is a more general one.

Examination of the tonsillar secretion in tonsillitis, not alone for bacteria which may be absent, but also for toxins, would be of the highest importance in practice. Many cryptogenic infections with their frequent complications, being grouped at present under the general heading of "colds" or "rheumatic angina" might be better explained and properly treated. Unfortunately, these methods of examination are not yet within the scope of general practice and even the laboratory methods have still much to be desired.

One would imagine that visual examination of the inflamed tonsils would teach us much regarding the causative infection. That would be the case were the inflammatory appearance of the tonsils different in different infections diseases. All that can be distinguished by macroscopic examination is the severity of the inflammation. It is probable that this severity runs parallel to the toxicity of the lymph. It is only in this respect that we can derive any information from the gross aspect of the tonsils. But even diphtheria may, as shown by Meyer's bacteriological investigations, present the clinical picture of a simple lacunar tonsillitis. Conversely, an apparently simple infection may present a most virulent clinical picture. Consequently, all we can do is to diagnose infection of the tonsils and no more; other symptoms must indicate what the underlying infectious disease is. Histological examination only shows the picture of inflamed adenoid tissue—no matter what the stage of the inflammation or the severity of its course. The characteristic finding in the microscopic examination is, as pointed out by B. Fränkel in 1896, an increase in the emigration of leucocytes from the tonsils.

In tonsillitis we have the clinical signs of inflammation—redness, heat, pain and swelling. In addition, also, we find a symptom—dryness of the throat—which points to an interference with the physiological function of the tonsil. This dryness of the throat is evident on inspection. It is hard to imagine that a diminished flow of lymph to the tonsil is the cause of such disturbed function.

The swelling of the tonsil, which cannot entirely be attributed to increased blood content, would tend to indicate an increased lymph content. Again, the increased emigration of leucocytes from the follicles would point to the same thing. The only conclusion is that there is an interference with the outflow of lymph from the tonsils, due to edema of the mucous membrane of the excretory ducts. Where there is a hematogenous etiology for the tonsillitis it is quite natural that the redness be present wherever adenoid tissue exists. In such case the tonsil inflammation is only a part symptom of a condition which may be termed *angina erythematosa*.

The initial stage may be of very short duration. General infections of the mildest type may, after a brief existence, entirely subside and disappear so that the lymph flowing to the tonsils again becomes normal. With the disappearance of the causative factor there follows an arrest and then a retrogression of the tonsillar inflammation. There is a return on the part of the mucous membrane of the status quo ante. Many cases of transitory pharyngeal pain and discomfort, after which the tonsils return to a normal condition, may thus be explained.

Local treatment in tonsillitis, once the initial stage has passed, can only be palliative. In acute inflammation, rest is one of the best remedies. We must try to ascertain the general condition giving rise to the tonsillitis, for angina erythematosa may be present in a severe disease as well as in a mild one. Where there is any suspicion of diphtheria, diphtheritic antitoxin should at once be administered. Otherwise, in the first instance, we must think of a rheumatic infection and this calls for salicylates. Ichthyol internally still deserves consideration as an antirheumatic. But we must always bear in mind in the treatment of angina, that we are dealing with an infection against which gargles are of little use.

Often the tonsillitis does not subside in the initial stage. In such an event we must suppose that the general infection is of longer duration or that the inflammation, once established, cannot subside immediately. The process, therefore, makes further progress and one of the first symptoms to be noted is disappearance of the dryness in the pharnyx. Inspection will also show a certain moisture of the pharnyx; but this does not make itself evident by a general gloss, as it does normally, but in the form of drops issuing from the lacunar orifices. Apparently the obstruction to the outflow of the tonsil secretion must undergo relief, or else there may be a subsidence of the inflammatory swelling of the lacunae or an increase in the pressure to the secretion to allow some of it to pass through. The latter possibility is the least likely.

Why does this tonsil secretion appear in the form of drops instead of spreading out in the form of a thin layer?

If the drops on the tonsil surface be touched with an applicator they will be found rather stringy in consistency, a property which is not shown by normal lymph. Because of this stringy quality the drops from the different lacunae may coalesce with the formation of a whitish secretion which may cover most of the tonsil surface and quite firmly adhere to it. This is not a membrane for it may be removed without injury to the underlying surface and it does not contain any fibrin. It consists principally of numerous cells, mostly leucocytes or epithelial cells. In addition to the cells large numbers of micro-organisms, mostly cocci, may be present, but no fibrin. In short, this secretion is of a mucous type. In this stage of tonsillitis the normal watery secretion of the tonsils is converted into a mucous secretion. Consequently, the tonsils, when inflamed, behave like mucous membranes and show catarrhal inflammation which is found not only on the tonsils but in all parts of the pharyngeal mucous membrane where adenoid tissue is present. Thus, we are dealing with an angina again which, in this second stage of the inflammation, may be termed *angina catarrhalis.*

In the first instance, treatment must be directed against the causative infection. Little is to be expected from local treatment so long as infected or toxic lymph circulates through the tonsils. While fever or other general symptoms are present we can only confine ourselves to palliative treatment. At any rate, the patient ought not to be disturbed too much with gargling as it has no effect. Sometimes no gargling is better.

Ordinarily, upon the subsidence of the general condition, the tonsillar symptoms will also subside. However, certain local after effects may occur. There may be, for example, a subacute catarrh with a moderate inflammation of the tonsils. In such a case local treatment with iodin is indicated. Applications of tincture of iodin give good results. The internal administration of iodin may also be of service. It produces a more watery secretion which irrigates the adenoid tissue, washing away all the mucus masses in the ducts, with their contained pathogenic and non-pathogenic bacteria. Gargles may now be of some similar service in mechanically removing adherent secretion. Ordinarily the transudation is of sufficient force to wash out the mucous secretion.

Usually the tonsils return to their normal condition. But not always. Sometimes the inflammatory process still continues. Fränkel states: "I do not deny that even in a simple lacunar tonsillitis the inflammation may, under circumstances, so increase as

to lead to an exudation of fibrin and to formation of membrane." Where the inflammation continues to the point of fibrin exudation the catarrhal stage of the tonsillitis passes into a fibrinous stage. This is true not alone for the tonsils but for adenoid tissue in general. Accordingly, we are again dealing with an angina which we may term *angina fibrinosa*. By this designation, we mean the third stage of inflammation of the adenoid tissue in the pharnyx. We need not sharply differentiate, as Fränkel does, this form of angina from angina catarrhalis or lacunaris. He seems to regard angina fibrinosa as an infectious disease *sui generis*. The fact is, these anginae are merely different stages of the same disease-process. One may merge into the other. The writer's interpretation of it is that the fibrin formation is not dependent upon the kind of infection but on the severity of the inflammation. This explains Meyer's observations on diphtheria, which undoubtedly gives rise to an angina fibrinosa, in that he met cases giving a clinical picture simply of a catarrhal inflammation of the tonsils.

Consequently, we must assume that the fibrinous stage always follows the catarrhal stage. True, this is generally the case; but the inflammation may be so sudden in onset that clinically the catarrhal stage seems to be absent, the fibrinous stage apparently making its appearance at once.

Obviously, in angina fibrinosa the lymph is so changed pathologically that it coagulates immediately upon its passage through the adenoid tissue. Probably it is the exposure to the air which brings about this coagulation—not that coagulation of the lymph on contact with the air is in itself pathological. Normal lymph will also coagulate, but not immediately. Immediate coagulation is pathological. When the coagulability of the lymph is raised to a certain point it shows itself by the presence of an angina fibrinosa. It has already been pointed out that this will first appear on the tonsils because here the conditions are most favorable for the retention of the coagulated material.

All that serology teaches concerning the coagulation of lymph under various conditions may be of importance for the understanding of the clinical value of angina fibrinosa. We know that different bacteria, as well as toxins, are able to increase the coagulability of lymph. Unfortunately, it is still impossible to obtain human lymph for laboratory experimentation. Perhaps by means of a modified blood suction apparatus, as described by Spiess, it may be possible to obtain such lymph from the tonsils for experimental purposes.

Clinically, we are accustomed to regard with gravity the presence of fibrinous exudate on the tonsils. Experience teaches that

a tonsillitis which reaches the fibrinous stage is, as a rule, indicative of a severe infection. This agrees with our supposition that the fibrin formation follows the greater toxicity of the lymph. But we must bear in mind that the same bacteria may produce infections of different degrees of severity. The diphtheria baccillus may in one case produce nothing more than a catarrhal angina, while an apparently harmless staphylococcus may in another case give rise to a most virulent infection. A severe infection of short duration is less to be feared than one of milder symptoms but of long duration.

There is a tendency to judge the kind of disease by the size of the pseudomembrane on the tonsils. It is true that diphtheria often spreads by the formation of a large membranous fibrin coagulum, but cases where this is absent are not infrequent. The size of the pseudomembrane is of no definite significance. Just as it may happen that the drops of lymph emerging from the lacunar orifices may become confluent, the confluence being influenced by various conditions independent of the properties of the transudation fluid, so it is also possible that in heightened coagulability of the lymph, the lymph in the lacunae may form such large coagulated masses as to block a further exudation of the fluid and its extension over the tonsillar surface.

This brings up still another point, namely, that the coagulability of the lymph may be so increased that its exposure to the air, to bring about coagulation, may not at all be necessary. That is to say, the coagulation takes place, in such an event, in the tonsillar tissue (follicles), the little whitish masses passing through the mucous membrane and giving rise to the clinical picture of follicular tonsillitis.

There may be also a still greater degree of tissue inflammation than simple fibrin formation in the lacunae. We may have a fourth stage of inflammation of adenoid tissue in which there may occur pus formation ending in ulceration—*angina phlegmonosa.*

It is presumable that every inflammation of adenoid tissue when it overreaches a certain point, becomes pustular. That is only possible under a variety of causes independent of the kind of infection. Inasmuch as the degree of the tonsillar inflammation is proportional to the toxicity of the lymph passing through the tonsils, a phlegmonous angina would occur with a most virulent infection. But it should be observed again that the dangerousness of an infection depends not only upon its severity but also upon its duration.

In the presence of a fibrinous or phelgmonous angina our first duty is to determine the kind of infection for this specific diagnosis will direct our therapy. Where it is one against which we have a specific remedy, as in diphtheria, naturally we are to use it at once. Where we have no specific remedy against the infection we are thrown again on our general means of combatting infections, such as salicylates etc. As already stated, local treatment, so long as fever or other general symptoms are present, has little effect. The most powerful therapeusis must remain ineffective so long as the toxicity of the lymph passing through the adenoid tissue continues.

Abatement of the pharyngeal pain is usually coincident with the subsidence of the general infection but the inflammation of the adenoid tissue does not necessarily at once subside. As already indicated, subacute, more or less painful inflammatory symptoms may remain and cause discomfort for a considerable time. These symptoms are of local character, assuming, of course, that the circulating lymph has returned to a normal constitution. There may be some mechanical obstruction, such as swelling of the ducts or the presence of coagulated fibrin masses in the ducts, preventing a return of the tonsil to normal function. For the swelling, applications of iodin or of astringents, or gargling with astringent solutions, are of service. Coagulated fibrin masses located superficially may thus be removed also, but those situated deeper will in time be washed out by the normal force of the lymph stream. But as this takes time, the patient in the meantime becomes a baccillus carrier, especially in cases where these fibrin masses serve as a good nutritive ground for the multiplication of bacteria. It should be accepted as a rule that any infectious disease associated with a fibrinous or phlegmonous angina will retain fibrin masses lodging the specific bacteria. Whether this will be for a longer or a shorter time cannot be stated in advance; hence it is advisable to regard all such patients as bacillus carriers and to treat them as such. To eradicate all these masses mechanically is, of course, out of the question. A general washing out by means of the lymph stream will serve the purpose. Accordingly, I make it a rule, after every case of fibrinous or phlegmonous angina, to place the patient for a few days on several grams of potassium iodid in order to bring about a more powerful watery secretion through the adenoid tissue. Furthermore, I employ the tonsil presser to squeeze out these fibrin masses. For the pharnygeal tonsil I use my catheter-like nasal applicator with which I press out the entire naso-pharyngeal

cavity. Iodin or silver solution may be applied after this procedure.

Chronic tonsillitis I ascribe to a lymph stasis as a result of plugging of the excretory ducts. The irregular enlargement of the tonsils so frequently encountered is due to the occlusion of some of the ducts. This occlusion is more easily explained by the retention of fibrin masses in the lacunae than by inflammatory swelling of the mucous membrane, although it is not my desire to try to exclude such inflammatory swelling in chronic tonsillitis. To remove the fibrin masses in these cases it is even necessary to go further and open up the lacunae and make a free exit for the lymph stream. Of course, where the adenoid tissue is already degenerated and the lymph transudation has been entirely lost such a procedure is out of the question. It should only be employed where the consistency and appearance of the enlarged tonsils would warrant an expectation of a return to normal. To expect this to occur with hard, fibrous, degenerated organs is not to be thought of.

Where the degeneration of the tonsil tissue makes a return to normal function impossible, they can only be regarded as tumors and the question of extirpation taken into consideration. Depending upon our viewpoint as to these tumors hangs the question of approach. If we consider them merely benign they should be removed only insofar as they are obstructive. This will depend upon the amount of space they encroach upon, their interference with breathing, tone production and swallowing. For such cases tonsillotomy is all that is required. A stump is left which is equivalent to a normal tonsil. The space relations are then quite normal and the only difference is that no lymph passes through a stump.

In America, particularly, the tendency has been to remove every vestige of tonsil tissue. Such practice can only follow the view that the degenerated tonsil tissue possesses some dangerous potentiality, that it is not entirely benign, or that the presence of the tonsil tissue offers a portal of entry for pathogenic germs into the system, with infections resulting therefrom.

Is this view justified?

It is claimed that the diseased tonsils often show an exacerbation of a chronic inflammation with symptoms of general infection. But as we have already seen, normal tonsils will also show this inflammation, which is attributable to the passage of pathological lymph through them. If healthy tonsils show this reaction to toxicity, inflamed tonsils will show it even more readily. It is only in a tonsil that has undergone complete fibrosis that no fluid will pass through.

Fibrous hardening of a whole tonsil may occur. It is just in such cases that less pharyngeal pain is complained of. In the course of an infectious disease such a tonsil will be found either not at all or only insignificantly inflamed. In the majority of cases, however, the hardening of the tonsil is incomplete and irregular in distribution, so that relatively healthy portions of the tonsil alternate with more or less completely fibrous, degenerated portions. The difference in consistency of the various portions of the tonsil may be ascertained by a probe. During an exacerbation, such a tonsil will also show difference in color and degree of swelling. The most normal parts are the ones to show the greatest swelling and redness.

Assuming that the pain and other discomforts in an inflammation of such a partially fibrous tonsil are greater than in an inflammation of a normal tonsil, it does not prove the tonsil guilty of the general infection. It must be proved that general infections occur oftener from diseased tonsils than from healthy tonsils. It must be admitted, however, that where the tonsil is diseased the conditions for infection from the pharnyx are more favorable than in healthy tonsils because of the absence of the protective action of the lymph flow. On the other hand, complete fibrous hardening is the end-result of degeneration of tonsil tissue and such condensation and hardening would tend to prevent the entrance of germs from the pharnyx into the tonsil, and as the lymph stream is in stasis, entrance of infection by this route is also unlikely. Consequently, a tonsillitis under such conditions would be strictly isolated. A systematic extension of infection would be possible only if the infection could reach the capillaries behind the tonsil fossa. And even then, the infection might be confined to the peritonsillar tissue.

Bacteriologically, the question has not been settled. True, we find pathogenic bacteria in diseased tonsils; but we also find them in other parts of the body. The question can always be asked: Did the tonsils infect the system, or did the system infect the tonsils?

Extirpation of the tonsil will, of course, remove the discomfort associated with their acute inflammation, but that is purely a local proposition and the operation is but of local use, with which we should be satisfied perhaps. But so far as the avoidance of general infection is concerned as a result of removal of tonsils we should put such thoughts aside. The fear that if, in the course of a tonsil enucleation, some tonsil tissue is left behind which may

serve a source of general infection, plays no importance at all from the standpoint of the views here advanced.

Conditions are different, however, where a tonsil inflammation is complicated by a peritonsillar abscess. In such an event we must assume that the infection was derived from the pharnyx through the tonsil. The tonsil, then, is the guilty organ and as we know that peritonsillar abscess tends to recur if the tonsil is spared, it should be removed, even though it have an apparently normal appearance.

I am in the habit in every case of peritonsillitis to remove the upper portion of the tonsil for most of the inflammations originate from the upper pole. Such removal also affords better drainage and it is more certain than incision of the anterior pillar. I do not wait until there is fluctuation but operate when there is some swelling and redness of the peritonsillar region. The origin of the peritonsillar abscess is in the lymphatics running laterally to the tonsil, and it is only after breaking through that the pus can drain through the usual incision. With my practice to remove the upper pole of the tonsil I reach the infected area directly and mere insertion of a probe will open up the abscess and the space in the supratonsillar fossa affords better drainage. I employ a Trautmann adenoid curette which can easily be inserted over the superior pole of the tonsil and by exerting pressure a good sized piece of the tonsil up to the hilus can be amputated. This operation of partial tonsillectomy is not identical with tonsillotomy amputation because the tissue is amputated up to the hilus, which is not the case in a tonsillotomy.

To sum up, we find that viewing the tonsils as lymphatic transudation glands throws a great deal of light upon their pathology. The tendency has been to regard the tonsils as useless organs, notwithstanding that we did not know what their function is. No organ should be condemned merely because we do not know what purpose it serves. I have said nothing regarding the practice of American operators in their tonsillectomy to also remove the capsule of the tonsil—a procedure that has any reason only if we consider the tonsils as malignant tumors. This procedure will shortly condemn itself (in the July 1916 number of The Laryngoscope cases of lung abscess following tonsillectomy are described). But even an intracapsular tonsillectomy, although an operative method that may be justified, is only to be employed when, upon scientific grounds, a death sentence against an otherwise useful organ must be pronounced.

ABNORMAL VOICES: FALSETTO, NASALITY, HOARSENESS. CLEFT PALATE SPEECH, CHOREATIC SPEECH, ANARTHRIA, THE VOICE OF THE DEAF AND THE MENTAL DEFICIENT.

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Voice—the audible sound produced in the larynx, modified in the passage outward through the pharynx and mouth so as to produce speaking and singing, in which the whole respiratory apparatus is concerned—is often interfered with from both physical and psychological causes. When the lungs fail to force a column of air, under varying degrees of pressure, through the trachea or windpipe against the vibrating portions of the larynx there will arise defects in phonation. On attempting phonation the moveable cartilages to which are attached the posterior ends of the cords (arytenoids) are seen to raise themselves in the fold of mucous membrane which covers them and rapidly approach each other. In addition to these movements the vibrating portions of the cords can be shortened or lengthened or limited to their edges; the free edges can be sharpened or blunted and the whole cord can be flattened out like a band. The entire mechanism is adapted to meet varying degrees of pressure of the column of air in the trachea. Thus in the falsetto voice we find only the edges of the vocal cords vibrating. In dyspnoea we find the degree of air pressure less than for the normal voice, this arising from the lack of breath or difficult breathing popularly known as shortness of breath due either to lack of oxygen, to excess of carbon dioxide or to the presence in the blood of certain products of muscular activity. The condition may be either inspiratory or expiratory or both and the chief causes are obstruction of the nose, throat, larynx, trachea and bronchi, etc. It is also a symptom of anemia and paralysis of the muscles of respiration.

Tone has three attributes; strength or loudness, pitch and quality or timbre. Strength depends upon the energy of the respiratory blast and upon the structure and adjustment of the vocal organs and the resonance apparatus. Pitch depends upon the rapidity of the vibration, this being determined by the length and tension of the vocal cords. Quality depends upon the character and number of the overtones accompanying any fundamental note.

In the abnormal voice some one of these elements is somewhere interfered with. In aphonia and hoarseness there may be paralysis

of the respiratory muscles or it may be caused by diseases of the larynx or by pressure on the larynx by abscesses or any kind of morbid growth or it may be traced to some functional or organic disturbance of the vocal cords. Aphonia is also very commonly associated with hysteria. In anarthria there is a disturbance of pronunciation due to a lesion between the cortex and the nuclei for the muscles of speech; it is always accompanied by disturbances of the other functions of other muscles than those of speech. In aphasia we have an exclusive disturbance of speech due as we have seen to a motor or sensory disturbance. In choreatic speech there are involuntary muscular movements—otherwise usually shown externally by twitchings of the face and limbs affecting the muscles of the larynx.

The principle difference between male and female voices lies in their pitch; they are also distinguished by their quality, the female voice being softer.

The high-pitched falsetto or eunuchoid voice, which occasionally occurs in men otherwise normal, caused by the extreme edges of the vocal cords alone vibrating, can be corrected by a course of vocal gymnastics and singing exercises, beginning with a note of high pitch and then singing down the scale until the lowest possible note on the register is reached. The voice is then kept at this pitch, and the low notes are repeated ten or fifteen times. Reading aloud in these deep tones secured by singing his way down the scale; training the patient to lower the position of the larynx during phonation; producing series of grunts, groans or growls; practicing all vowel sounds upon the "direct attack" (coup de glot); using each vowel as an exercise for crescendo and diminuendo; whispering exercises; megaphone exercises; phrasing, linking, inflection exercises are all to be practiced regularly. Further aid may be gained from exercises that will strengthen resonance; such as: sing steadily on one note, *hung*, making *ng* last during three counts; sing steadily on one note, hung, ee, as long as breath will last; sing steadily on one note, hung, ay, as long as breath will last; sing steadily on one note, hung, ah, as long as breath will last; sing steadily on one note, hung, aw, as long as breath will last; sing steadily on one note, hung, oh, as long as breath will last; sing steadily on one note, hung, oo, as long as breath will last. Say each vowel sound-staccato, six times; say each vowel-sound faint voice, six times; say each vowel-sound loud voice, six times. Chant: hung on a tree, hung in a closet, hung by a rope, hung at the top.

Whistling, blowing out candles, singing arpeggios downward on vowels, feeling larynx move down during the progress from ee to oo, placing b's and m's before the vowel sounds and forcing them out repeatedly on the lowest tone gained by practice are also good exercises to be followed for the deepening of this abnormal voice.

A knowledge of the few following facts will suffice to correct the habit of nasalizing vowels. The soft palate which hangs at the back of the mouth acts as a valve on the passage of the nose. When the top of the soft palate is arched backward from its point of junction with the hard palate, it covers the internal nasal aperture, and the breath passes altogether through the mouth. When the soft palate is relaxed and pendant from the edge of the hard palate, the breath passes partly through the nose and partly through the mouth, and when the mouth passage is closed (by means of the back of the tongue, as in *ng*; or by the forepart of the tongue as in *n*; or by the lips as in *m*) the breath passes out altogether by the nose.

The chief difficulty lies in the recognition by the ear of pure oral and mixed nasal quality. The action of the soft palate may be seen by opening the mouth. Then, by pressing the top of the soft palate with the thumb, or with the india rubber end of a lead pencil, the internal nasal aperture will be discovered, and the utterance of "ah" and "aw" will be purely oral. Repeat these vowels with and without the mechanical pressure, and after a few experiments the ear will distinguish the difference between oral and nasal sounds. Practic on other vowels, in forming which the soft palate cannot be seen, will soon develop a feeling of the difference.

But the readiest way to gain a perception of the denasalizing action of the soft palate will be by the following exercises:

Sound the consonants *m, b* without separating the lips, as in the word "ember." The change from *m* to *b* is nothing more than the covering of the nasal aperture by the soft palate: and the change from *b* to *m*, without separating the lips, as in the word "submit," is merely the uncovering of the nasal aperture. The tendency to nasalize vowels is most felt when they occur immediately after or before nasal consonants—*m, n, ng*—but many persons nasalize every vowel.

The French elements "an, en, in, on, un, am, em, etc.," are merely nasalized vowels. The great misunderstanding that prevails about nasal speech might right here be corrected. It is only the habit of negligently allowing the uvula to be inactive when producing the *m, n,* or *ng* that is objected to the closing of the nasal

passage altogether is not at all necessary, for then we have a harsh, nonresonant tone.

A voice that is harsh or rough, as from a cold or from fatigue, may, of course, simply need medication and rest; but if a chronic hoarseness exist, a state of throat that is often the result of continued poor breathing habits as well as the effects of hysteria, exercises in singing and speaking *ah* with the glottal catch at the beginning and end of each sound may be practiced; the breath held back by closing the glottis: the vowel begins suddenly with strong vibrations, it is ended by snapping the glottis shut again.

For stiff throat think above it for resonance, below it for breath —in fact any distraction from the larynx will relax it.

A more serious organic cause of defective speech is cleft palate, where an opening exists between the mouth and the nasal passage. The breath which requires to be shut within the mouth for p-b, t-d, k-g, escapes by the nose, and a percussive articulation is impossible. In most cases a skillful dentist can cover the fissure in the palate by a suction plate, and thus enunciation may be gained. A cleft palate causes all vowels to be nasalized and as corrective exercises for this, first have each vowel practiced religiously while holding the nostrils tight shut with the thumb and first finger. Frequently this nasalizing of the vowel is mere habit, without any organic cause. Surgical measures give the best results in cases of cleft palate and palate perforations, but certain specially made plates called Protheses will often produce a remarkable improvement in the speech. In the case of palatal paralyses electricity and exercises will give the most benefit.

R. Tait McKenzie, in "Exercise in Education and Medicine" (pp. 539-545) says: "There are certain functional disturbances of the nervous system that show themselves in spasmodic habit movements of the face and body, like blinking and torticollis, in disordered co-ordination of speech like stuttering and stammering, and in choreicform movements of the body.

They may be classified under the general term *tic* as are all psychoneuroses, often varying directly with fatigue and partly restrained by the will. They are physiological acts that have become meaningless through inco-ordination and pernicious habit. The treatment must be re-education of co-ordinate movements and the training of the patient to break up the harmful neuromuscular custom.

The inhibition gymnastics of Oppenheim lay great stress on training to voluntarily *inhibit a reflex*. A pointed instrument is ap-

proached to the eye of a blinking patient, who is admonished not to blink. The patient is touched and instructed not to start away as formerly. The exercises *aim to train the will and control.* In his exercises Brissand tries to replace abnormal by normal movements, and especially to train the patient in immobility where they have an uncontrollable desire to move, just as does Oppenheim. He gives them treatments three times a day, each one short enough to avoid fatigue and to maintain the interest of the patient. In these exercises the antagonistic movements should be used; for example, drawing the mouth to the left where the habit is to twitch it to the right. Slow, deep breathing with the back to the wall, arms raising and lowering, as in Figs. 406, 407, Pitre's method, makes a good introduction to a treatment, while sports and games demanding attention, skill and accuracy are of marked value. Under treatment the prognosis is good, except in defective subjects.

Certain forms of tic require special treatment. Charles K. Mills treats aphasia in a purely pedagogic way by repetition of letters, words, phrases, and sentences recognized by the patient in reading or after seeing or touching."

Chorea is a disturbance of co-ordination characterized by regular involuntary contractions of the muscles, accompanied by psychic disturbances and frequently by endocarditis. It occurs especially in abnormally bright, active minded children who are forced ahead of their grade in school and are subject to the excitement of competition for prizes. Girls are three times as liable as boys. Will S. Monroe sent an inquiry to twenty-one teachers, thirteen of whom had one or more cases in their schools. Of these twenty-four cases sixteen were girls.

While there are no constant anatomic changes found in the nervous system, the tendency to endocarditis is such that in 110 cases of autopsy the effects were noted in nearly 100. Because of this great liability to endocarditis the heart should be carefully examined in all such cases. The presence of a murmur, however, does not indicate endocarditis, since the disease occurs most frequently in nervous young girls in whom heart murmurs are almost the rule. The duration of an attack is variable, from two to three weeks or the same number of months, with an average of about eight or ten weeks.

The disease is easily spread by psychic contagion, hence the necessity for early diagnosis and isolation.

Every choreic child passes her novitiate of restlessness, muscular twitchings of the arms, shoulders, legs, awkwardness in the execu-

tion of delicate tasks, exaggerated movements of the face muscles, wrinkling of the forehead, and knitting of the eyebrows, early evidences that are apt to be overlooked or misunderstood.

The treatment of the acute attack consists in rest, isolation, and tonics. Where the jerky movements have abated the application of general massage is of value, and the importance of gymnastic exercises including mouth gymnastics and vocalization exercises cannot be overestimated during the convalescence. When the acuteness of the attack has subsided, the treatment should begin with massage for the first few days, followed by simple, slow, passive or resisted movements, in which the limb is held quiet for a few moments, and the rhythmic passive movements in which the patient takes no active part. The patient will oppose these movements by the incoordinate jerky movements due to the disease. Later on, rhythmic exercises sufficiently active to tire the muscular system moderately will be in order. These should be done with the patient by himself or herself, if possible, and any excitement or competition of class drills should be seduously avoided. If the exercises are taken in company with others, the patient should be placed in the back row of the class.

The keynote of such treatment is the rhythmic repetition of simple movements to overcome the irregularity of the twitching, and great strain on the attention or fatigue of the patient should be eschewed. The practice of simple dancing exercises to music is of the utmost service, emphasizing the rhythm and taking away the mental strain necessary to follow movements done to command.

Dr. E. B. Huey* says in his "Backward and Feeble-Minded Children" that the public schools receive and partially control, for a time, almost all of the individuals who will later trouble society as delinquents or dependents, or who will be troubled themselves by insanity or other forms of mental disturbances. Usually only the lowest grade of feeble-minded children fail to find their way to school. He also says among other statements:

Of even greater importance, we may find, will be the early diagnosis of dangerous mental tendencies and habits, that occur even in the brightest and best of school children. The easing of adaptations in critical directions and periods, judicious counsel to parents and others who may direct the child toward such levels of occupation and environment as will be safest and most productive for him —these are services which the history of cases of insanity teach us may be rendered to thousands of threatened lives. The schools

*Huey, Edmund Burke: "Backward and Feeble-Minded Children." Warwick & York, Inc., Baltimore, 1912.

and institutions are already beginning to look for men competent to do this work, and psychology and medicine are just beginning to realize that they must join hands with each other and with sociology and education in training clinicians capable of rendering this service.

Dr. Huey's remarks have suggested to me the great possibilities before us in this field of speech correction. Most all mentally defective children are afflicted with negligent speech. Why not work along these lines of corrective speech exercises to do some character building? Breathing exercises, mouth and tongue gymnastics, vocal exercises in rhythm in connection with physical exercises, training in association of ideas, in reaction time, in self-ocntrol, in accurate attention, in correct bodily poise, in clear enunciation and in fine, all the manifold applications of the work we are doing for the speech defective, in order to have him overcome his physical or psychic disturbance.

By deaf-mutism we understand a condition of deafness, either total or partial, followed by failure to learn to speak or by loss of speech already learned. The inability to speak is the result of the deafness and not of any defect of the speech mechanism. *Congenital* deaf-mutism is attributed mainly to heredity and consanguinity, also to alcoholism, syphilis, difference in the ages of the parents, and to diseases of pregnancy. *Acquired* deaf-mutism is due chiefly to meningitis. It also frequently follows scarlet fever. Other diseases, like measles, diphtheria, typhoid fever, mumps, influenza, etc., sometimes produce it.

What is to be done with the deaf-mutes? The one thing of supreme importance is to teach them to speak. Mental awakening is the *first* step in the training. The mind of the deaf-mute is often in a backward condition because he does not fit with his surroundings. Exercises should include mimic movements, following the teacher in running, walking, sitting, blowing feather, etc. Games (such as catching ball, tossing beanbag, hiding thimble, etc.) should be attempted. Thus the child's attention, observation, imitation, and imitative construction are developed.

The speech development proceeds along the following lines:

1. Breathing through the nose and mouth are first taught. The teacher breathes through the nose on a slate or a mirror and shows the two moist spots; the child learns to imitate this. The mouth breathing produces one spot.

2. The low position of the tongue is necessary for proper speech. It is taught by showing the position and using the mirror, and by breath exercise. This latter rests upon the fact that the child

cannot produce a good-sized spot on the slate unless he keeps his tongue down.

3. Tongue gymnastics are next used to limber up and train the muscles which have never received the proper development. The tongue is protruded, retracted, moved to each side, turned up, etc.

4. Tongue training preparatory to various consonant sounds is introduced. For example, the child is taught to put the tip of the tongue against the upper teeth; this gets him ready for the letter "t." He learns to draw it back and close it against the hard palate, thus getting ready for "k."

5. Vibration of the vocal cords is taught by feeling. The pupil puts his hand on the teacher's chest and also on his own. He thus learns to make a tone. He learns to raise and lower the voice, and by careful drill is able to make a fairly good tone.

6. The physiological alphabet consists of a set of diagrams giving the typical position of the tongue and lips for the chief sounds of the language. Here for example are the diagrams for "t" and "k." The child learns these diagrams just as we learn the alphabet, so that he can both make the sound and write the letter or group of letters for it.

7. Combinations of consonants and vowels are now read at sight. Through these combinations words and sentences are developed.

8. Lip-reading of words and sentences are taught by having the patient watch the teacher's lips while she distinctly enunciates some word. Thus he learns to pick up objects off the table, to point out parts of the body, to obey commands, etc.

When deafness is acquired after the person has learned to speak, the teaching of lip-reading should begin at once. The voice then retains its natural character and the person can go right along with his education.

The unnatural character of the voice which the congenital deaf-mute acquires is the one drawback; still it is immensely better to be able to understand well and to speak fairly well than not to be able to do either. There is, however, a series of methods recently developed by the science of experimental phonetics by which the niceties of pronunciation are studied and taught. Foreigners are also enabled by these methods to acquire a correctness of pronunciation otherwise impossible. The person is taught exactly how to place his tongue, to produce his nasal breathing, to regulate the length and intensity of his vowels and consonants. Here the work of special private instruction must begin for those deaf-mutes who wish more than the schools can offer.

55 Central Park West.

LATERAL SINUS THROMBOSIS. THREE CASES.*

Dr. David H. Ballon, Montreal, Canada.

In this paper I give the history of three cases of lateral sinus thrombosis which I successfully operated on at the Royal Victoria Hospital, Montreal.

Case 1. J. O. Male, age, 15. August 31, 1912. Ten years ago the patient had measles, and since then he has had a painless purulent discharge from the right ear off and on, associated with deafness. These were the only symptoms, and they inconvenienced him very little.

Five weeks ago while crossing the Atlantic he contracted a cold, the right ear began discharging and kept up until two weeks ago, when it suddenly ceased, and then he began to suffer severe pain in the same ear, radiating over the whole right half of the head and back. The right side of the neck was stiff. The mastoid was tender but not swollen.

During the last week he has had daily chills of at least five minutes' duration, intermittent fever ranging from the normal to 101°, and sweats, and during the last three days he has also been somewhat dizzy and has been vomiting. His eyesight is good, and there are no other complaints. I saw him in consultation, made the diagnosis of sinus thrombosis and he was admitted to the Royal Victoria Hospital on August 31, 1912.

On examination the patient looked toxic. His temperature was 101° and pulse 100. The right ear showed slight prolapse of the posterior wall of the external auditory canal; the drum was completely destroyed, and the tympanic cavity was quite dry and only filled with epithelial debris. He heard loud speech on contact and gave the usual tuning fork tests for chronic suppurative otitis media. The mastoid was tender over the three cardinal points but there was no swelling. The right sternomastoid and the muscles at the back of the neck were rigid; the right jugular vein was markedly tender. No thrombus was palpable. There was no Kernig or pathological reflexes. The leucocyte count was 30,000. The

*Read before the section of Oto-Laryngology and Ophthalmology at the forty-eighth annual meeting of the Canadian Medical Association, Montreal, June 15, 1917.

blood culture was negative. The fundi were normal as reported by Dr. Byers. Medical examination of the heart, lungs and kidney showed no evidence of any septic focus. The x-ray showed a small sclerosed right mastoid with the lateral sinus far forward, but no thrombus could be diagnosed. Apart from a few pneumatic spaces the left mastoid was identical. The left ear was normal.

During the night the patient had a rigor, the temperature rising to 103.2° and the pulse to 130. Dr. Birkett saw the case and confirmed the diagnosis.

On Sunday, September 1, I performed a radical mastoid operation. The essential findings were as follows: After removing the sclerosed, ivory-like cortex pus welled up under tension. An abscess cavity was found filled with very fetid pus and cholesteatoma exposing the lateral sinus. The sinus wall was gangrenous; no pulsations were present; a thrombus was palpable. The sinus was then very freely exposed, removing the bone almost as far as the torcular before healthy sinus was found. In a similar way the lower end was traced down the neck. The sinus was then opened. No bleeding occurred, the thrombus was removed and the incision extended upwards and backwards until free bleeding was obtained at the upper end, when it was packed with idoform gauze, which controlled it. The same procedure was carried out at the lower extremity. Having obtained free bleeding at both ends the jugular vein was not ligated.

B. pyocyaneus was found in the culture of the mastoid pus.

Sept. 3. The patient had a chill lasting ten minutes. Temperature 105°; pulse 112; respiration 48. The temperature dropped in four hours to 100°. The patient perspired freely and complained of occipital pain. His lungs were normal.

Sept. 18. Large deep-seated abscess was discovered at the back of the neck. Free and multiple incisions were made. About a cupful of creamy fetid pus was evacuated and adhesions were broken down. The cavity was found extending from the external occipital protuberance to the first dorsal, and curiously enough there was no visible connection with the mastoid. Free drainage was established: rubber tubes inserted and the wound packed with iodoform gauze. From now on there was an uninterrupted recovery. I did the plastic operation to close the mastoid wound on Oct. 9, and he was discharged from the hospital one week later. He could hear loud speech two and one-half yards away with his right ear. April 4, 1916, the patient is perfectly well.

Case 2. A. S. Tailor, age 23. On Feb. 17, 1916, I was called to see a young man who gave a history of a frequent painless discharge from the right ear during the last seventeen years. The ear would light up now and again, following a cold, become painful, then gradually subside. One week ago he became ill with influenza and then had another acute exacerbation of the old trouble, with very severe shooting pain in the same ear.

An examination showed a congested drum but no bulging, with a small round perforation about the middle of the posterior half. There was scarcely any discharge; but epithelial, debris visible through the perforation, made one suspicious of cholesteatoma. He had no mastoid tenderness. The temperature was 101°.

I did not see him again until eleven days later, when he gave the following history: During the last week he had rigors every second day lasting about fifteen minutes, with very high fever, especially at night, and profuse sweats. There was absolutely no pain or discharge from the ear. He had frontal headache, slight photophobia, felt weak and dizzy. He vomited daily independently of his meals.

The patient looked very toxic; there was a cyanotic tinge to his checks and a herpetic eruption on his upper lip. The skin was very moist.

The right drum was reddened but not bulging, and the perforation had healed over. There was no discharge or mastoid tenderness. He heard whispered speech at one yard, and gave the usual tuning fork tests for an otitis media. Apart from a slight rotary nystagmus to the left there was no evidence of any labyrinthitis as the caloric reaction was normal. Barany's pointing test was normal. There was no rigidity of the head or back; the eye grounds were negative, and the cerebrospinal fluid showed no evidence of meningitis. He had a leucocyte count of 12,000. The blood culture showed no organisms. No tenderness was present over the jugular vein. An examination by Dr. C. F. Martin eliminated the possibility of a septic focus anywhere else in the body. The left ear was normal.

He was admitted to the Royal Victoria Hospital with the diagnosis of a sinus thrombosis on Feb. 27, and the same day he had a rigor lasting one-half hour, the temperature rising to 103°.

A skiagram showed a sclerosed right mastoid and a pneumatic left mastoid.

The important findings at the mastoid operation on Feb. 28 are briefly as follows: The bone although sclerosed was extremely vascular. When the cortex was removed pulsating pus welled up.

The lateral sinus was fairly superficial, far forward and covered with granulations. A thrombus about 1½ inches long was palpable but moderate pulsations were transmitted. I exposed the sinus freely, tracing it back in the direction of the torcular and down the neck until healthy sinus was present. I then incised it, removed the thrombus and extended the incision first upward and then downward until free bleeding was obtained at both ends. The bleeding was controlled by packing with large rolls of iodoform gauze. The jugular vein was not ligated. The middle ear contained granulation tissue and a little cholesteatoma. March 5 I removed packing; no bleeding followed. There has been no pain or chills since the operation and the temperature has been normal. April 3, was discharged from the hospital cured.

Case 3. A. S. Female, age 20. Feb. 24, 1917. Six years ago following an attack of influenza patient's right ear began discharging and it has kept up more or less since. The discharge was fetid, moderate in quantity and unassociated with pain. During the last two months the patient has been quite nervous and hysterical at times; has had pain localized to the right ear, frontal headache, and occasional dizziness; but no vomiting, fever, chills or sweats. Her eyesight is fair. She has dyspnea on exertion and swollen ankles occasionally.

Examination showed a polyp completely filling up the right external auditory canal with foul pus oozing around it. She could hear loud speech three yards away. Dr. W. F. Hamilton reported a hemic systolic murmur in the pulmonic area; otherwise medical examination was negative.

On March 1, 1917, at the Royal Victoria Hospital, under general anesthesia I removed the polyp and granulation tissue which sprang from the attic and mastoid. The patient left the hospital next day in good condition; no pain or temperature.

A summary of events occuring during the following week are as follows: On her way home she caught a cold, had a chill and pain in right ear. She was, by the way, lately exposed to typhoid infection. During the week she had frequent and severe chills, ran very high temperature, often 104°, and perspired profusely. Her headache was mainly frontal, and localized to right half of the head and unrelieved by frequent doses of phenacetin. She was frequently delirious at night. She was very constipated. I saw her at midnight, March 10. She was very toxic; her temperature was 103.2° in spite of phenacetin powders, and her pulse 130. The right cheek

only was flushed, the tongue parched and furred. No nystagmus; there was slight edema over the lower part of the right mastoid. There was pulsation but scarcely any discharge from the ear. No photophobia or rigidity of the head; no tenderness over the jugular vein. Barany's pointing test was normal. She was readmitted to the Royal Victoria Hospital and the next morning I operated on the mastoid, found it sclerosed and extremely vascular. With the first chisel there was a gush of greenish-looking pus under tension, such as I had never seen before in mastoids. This pulsating pus kept welling up from the lateral sinus which was quite superficial and very far forward. The sinus was thickened, covered with lymph and granulations, and there was a greenish-yellow gangrenous area about the size of a five-cent piece. No thrombus was palpable. Pulsations were distinctly visible and palpable. Under these circumstances, I freely exposed the sinus until it appeared apparently healthy, but did not open it. The mastoid was small and filled with cholesteatoma and granulation tissue.

Another interesting feature of the operation is that while working along the tegmen there was a sudden discharge of greenish pus which I demonstrated to be coming through the dura from the temperosphenoidal region. I enlarged this opening and then packed the whole wound lightly, leaving it wide open.

The following day the patient had a rigor lasting half an hour and the temperature rose to 105°. The right cheek was flushed; no headache; no meningeal or labyrinthine symptoms. The wound was dressed, the gauze removed; pulsations in the sinus were still present. The bacteriological report of the pus was *streptococcus hemolytycus*. The leucocyte count was 16,000. The blood culture was negative; the eye grounds and the lumber puncture negative.

The next two days there were no chills, and the temperature was comparatively low but the patient looked septic. I noticed an appreciable diminution in the strength of the transmitted pulsations from the sinus and the gangrenous patch in the sinus appeared depressed. At this time the patient had another chill, with a temperature of 104°. No tenderness over the jugular vein. Mental condition good.

I then decided to explore the lateral sinus and middle fossa. Dr. White kindly assisted me. The dura of the middle fossa was exposed, but as it appeared healthy and no sinus was traceable from the mastoid I left it alone. Then the lateral sinus was exposed more freely and incised about 1½ inches. It was found completely thrombosed, the clot was removed but no free bleeding could be

obtained from either end. I was unable to continue the operation on account of the patient's poor condition, the pulse rising to 160 and the temperature to 105.2°.

Rectal salines with whiskey were ordered every four hours. The next day the patient was better. No complaints. Pulse 88; temperature 100°. The wound appeared clean; the plug was removed from the sinus; there was no bleeding.

March 18. Three days after last operation the patient had a chill lasting three minutes, and the temperature rose to nearly 104°. A general medical examination by Dr. Hamilton was negative. I operated again. Dr. Archibald first ligated the jugular vein. There was no thrombus present. I then traced back the lateral sinus to the torcular, removed the thrombus, got very free bleeding, then packed with iodoform gauze and carried out a similar procedure at the jugular bulb, where I also got free bleeding. I cut away the gangrenous wall of the sinus. The patient stood the operation well. Then for a week I gave her urotropin, one drachm daily, and treated the wound with Dakin's solution by Carrel's method. It worked admirably.

I placed three tubes in the wound, one at the torcular end, another at the bulbar end, and the third in the middle ear, and injected 10 c. c. of Dakin's solution every two hours, day and night.

March 20. Temperature rose to nearly 103°. Pulse 112; respirations 36; patient was somewhat irrational. No chills but free perspiration. Without an anesthetic I removed the plug from the torcular end. The bleeding was free so that I had to repack it. Then on removing the plug at the bulbar end I found a bulbar abscess. There was a gush of at least a teaspoonful of yellowish pus and it kept welling up with every pulsation. Here I inserted another Carrel tube and injected 5 c. c. Dakin's solution every two hours.

There was a sudaminous rash all over the body. Four days afterwards I removed the plug from the torcular end; no bleeding, only pulsations.

March 26. Temperature normal, the patient feeling fine. *March 29,* she is out of bed. *April 7,* discontinued Carrel treatment. Three days later I closed the wound and made a Y-plastic. Two weeks later the patient left the hospital. A month later she came in from the country. The ear was completely healed and she was feeling fine. Discharged cured.

Without attempting to draw conclusions from only three cases I might briefly summarize them:

The thrombus was on the right side in all cases. Ages 15, 23, 20, respectively; chronic history of aural suppuration, 10, 17 and 6 years. Immediate causes: "cold," influenza, or operation. Organisms: *B. pyocyaneus, staphylococcus* and *streptococcus pyogenes*, and *streptococcus hemolyticus.*

The symptoms were characteristic, showing the usual triad; chills, intermittent fever and sweats. Two of the patients while in the hospital had rigors lasting one-half hour. The highest temperatures were 103°, 103°, 105°. There was a marked flush of the right cheek only, *i. e.*, the side of the lesion.

The aural findings: Case 1. Drum destroyed, tympanic cavity dry; but filled with epithelial debris. No mastoid swelling.

Case 2. Small round perforation in middle of posterior half of drum, scanty discharge, later absolutely dry, no swelling of mastoid.

Case 3. Drum destroyed, tympanic cavity filled with granulation tissue; moderate discharge and pulsation visible. Edema over the mastoid.

The blood cultures and eye grounds were negative in all cases, also the lumbar puncture.

Leucocyte counts: 30,000, 12,000, 16,000.

X-ray showed small sclerosed mastoid, sinus far forward but no thrombus.

The findings at operation were in the main identical in all cases. The mastoid was sclerosed but vascular. Pulsating pus under tension was present. The lateral sinus was superficial, very far forward, gangrenous, or covered with lymph and granulations. Pulsations were visible or palpable even with presence of thrombus. Tympanic cavity filled with granulation-tissue and cholesteatoma.

The treatment was a radical mastoid operation. Removal of all diseased bone in contact with the sinus until apparently healthy sinus was reached in both directions, *i. e.*, towards the torcular and jugular vein.

Where the thrombus can be removed and free bleeding obtained at both ends the jugular vein need not be ligated. This was the treatment in two cases, and in the third the ligation of the jugular had no effect, as there was no thrombus in the jugular vein and free bleeding was obtained in the bulbar end after ligation.

269 Bishop Street.

AN ATTEMPT AT SIMPLIFICATION OF THE PHYSIOLOGY OF THE VESTIBULAR LABYRINTH.*

DR. ISAAC H. JONES, Philadelphia, Pa.

When Father Adam first turned to the right, the endolymph in his semicircular canals lagged behind, relatively to the left. His sight and muscle-sense informed him that he was turning to the right: consequently he came to interpret endolymph movement to the left to signify that he was turning to the right. He always turned *away from* the endolymph, and for this reason he recognized endolymph movement in one direction to mean that he was moving in the opposite direction. Each child born of Adam passes through the same experience—he learns to interpret impulses from the labyrinth, just as he comes to interpret stimuli received from the retina. Images on the retina are upside down, but in course of time the child learns to reverse this image in his consciousness and to realize that the external object is actually right side up. Similarly, through countless repetitions he comes to recognize that endolymph movement in one direction means that he is moving in the opposite direction.

When an individual moves past external objects, his *eyes* attempt to fix upon certain objects as they pass. He does this in the attempt to stabilize the sensorium. When traveling in a railroad train his eyes fix upon telegraph poles and other objects as they pass—consequently his eyes move in the direction of the passing object. Similarly, when he is turning to the right, his eyes, although closed, have been trained to move to the left in the direction of the external objects which are moving to the left.

Experimental stimulation of the ear, by turning in a revolving-chair or by douching with cold or hot water, produces an *artificial* movement of the endolymph. After an individual is turned to the right a sufficient number of times to cause the endolymph to catch up with the movement of the body, he feels that he is standing still if his eyes remain closed, whereas he is actually turning to the right. As there is no movement of the endolymph in relation to the hair-cells within the ear, his interpretation is that he himself is not moving. Now if the chair be stopped, the endolymph by its momentum continues to move to the right; he therefore feels that he is rotating

*Read before the American Otological Society at Atlantic City, May 30, 1917.

to the left, although he is actually sitting still. If the right ear be douched with cold water, the chilled endolymph moves downward to the right and the individual feels that he is falling to the left.

The purpose of this paper is not to deal with physiology for itself alone—the main reason for *simplicity* in this subject is that at the present hour there is practical need for the Barany tests. Probably the most important service that the otologist can give to the government—a service that is peculiarly his own—is in aviation. Perfect equilibration is accomplished through an harmonious co-operation of the eye, the muscle-sense—and most particularly the "balance-sense" of the ear. The ear function is of peculiar importance, in that the vestibular labyrinth has for its sole function the maintenance of balance. After impairment or loss of one of the senses responsible for equilibration, compensation may take place to a certain extent, provided that the individual is on "terra firma"—the tabetic may be taught to avail himself of the visual-sense and of the balance-sense of the ear in co-ordinating his movements. Similarly, the blind man is able to walk by the aid of a cane until deprived of the guidance of either the muscle-sense or the balance-sense of the ear. Deaf mutes, in whom the ear-sense is destroyed, are enabled to maintain their balance by means of sight and the muscle-sense, and develop inco-ordination only in the dark or in the water.

However, when the human being becomes a bird, as it were, he suddenly finds himself in an entirely new environment. In flying through the air, on what does the aviator rely in order to maintain his equilibrium and that of the aeroplane? Can he rely on his sight? Hardly, for when he is sailing through the clouds or in the dark, his eyes positively cannot give him the slightest information as to his position in space, not even whether he is right side up or upside down. As regards the muscle-sense, it is undoubtedly true that it plays a certain part; but when the aviator is seated on an unstable and rapidly moving machine, it is hardly conceivable that the weight of his body could determine and maintain his position in space merely by the sense of gravity. In order, therefore, to preserve that wonderful accuracy necessary in operating such a delicate mechanism as the flying machine, it is obvious that he relies primarily upon his vestibular labyrinth. It is easily conceivable that some of the unexplained accidents in aviating may be due to a concussion of the internal ear, produced by the rush of the air or by the decrease of the usual air pressure when at great heights. Since normal internal ears are such an important asset to the aviator, good prudence would suggest a

most careful examination of the degree of function of one's internal ears, before taking up flying as an occupation. The ear-tests furnish an exact and mathematical analysis not only of the function of the internal ear, but also of the entire vestibular apparatus. When a candidate for the aviation service of the government presents himself, it is for his own personal interest as well as for the good of the service, that every portion of his "balance apparatus" should be declared intact and normally functionating. This can be absolutely determined by the ear-tests,—"Yes" or "No." If, after the Barany tests, the candidate shows normal responses in nystagmus, past-pointing and falling, he is fit for the service. If he does *not*, he is unfit.

The following standard for entrance in the Aviation Section of the Signal Corps of the United States Army has been approved and is to constitute the requirement for admission into this service.

Equilibrium—vestibular tests.—The nystagmus, past-pointing and falling, after turning, are tested. The turning-chair must have a head-rest which will hold the head 30 degrees forward, a foot-rest and a stop-pedal.

(a) *Nystagmus.* Head forward 30 degrees; turn candidate to the right, eyes closed, 10 times in exactly 20 seconds. The instant the chair is stopped, click the stop-watch; candidate opens his eyes and looks straight ahead at some distant point. There should occur a horizontal nystagmus to the left of 26 seconds' duration. Candidate then closes his eyes and is turned to the left; there should occur a horizontal nystagmus to the right, of 26 seconds' duration. The variation of 10 seconds is allowable.

(b) *Pointing.* 1. Candidate closes eyes, sitting in chair facing examiner, touches examiner's finger, held in front of him, raises his arm to perpendicular position, lowers the arm and attempts to find the examiner's finger. First the right arm, then the left arm. The normal is always able to find the finger.

2. The pointing test is again repeated after turning to the right, 10 turns to 10 seconds. During the last turn, the stop-pedal is released and as the chair comes into position, it becomes locked. The right arm is tested, then the left, then the right, then the left, until candidate ceases to past-point.

3. Repeat pointing test after turning to the left.

(c) *Falling.* Candidate's head is inclined 90 degrees forward. Turn to the right, 5 turns in 10 seconds. On stopping, candidate

raises head and should fall to right. This tests the vertical semicircular canals. Turn to the left, head forward 90 degrees; on stopping, the candidate raises his head and should fall to the left.

Any candidate for service in any branch of the army or navy must conform to a physical standard. He must be a normal man, such as would satisfy the average insurance company, with the additional requirements of minimum height, weight, chest measurement and auditory and visual acuity. The aviator must have not only these requirements, but must also have a normal balance mechanism. Therefore, the determining test for the aviator is the Barany test.

Therefore the peculiar test, applied only to the aviator, is the Barany test.

Medical Arts Bldg.

PERIODIC ATTACKS OF VASOMOTOR RHINITIS FOLLOWED BY ASTHMA AND SYMPTOMS OF PARANOIA, RECURRING OVER A PERIOD OF TEN YEARS.

Dr. John R. Davies, Jr., Philadelphia, Pa.

This case is interesting in many ways, and the scarcity of the literature on the subject prompts me to report it.

The patient is a female, 51 years of age, unmarried, and passed the menopause about two years ago. There is no history in the family of any mental disease. She is the youngest of six children. There is nothing significant in her previous history, except that as a child she was most bad tempered.

The first attack of vasomotor rhinitis occurred about twenty years ago. While dancing, the patient became overheated, and then took a straw ride without a coat, and the next day had what she thought was a severe cold. This proved to be the first attack of vasomotor rhinitis and lasted about six weeks. At that time there were no asthmatic symptoms or unusual nervous manifestations. The following June she had her next attack, and this lasted until late in September. These attacks recurred annually over a period of nine years. They began in June and lasted the greater part of

the summer. During this period there were no nervous disturbances except the depression that is occasionally seen with hay fever and there were no asthmatic attacks.

About ten years ago the duration of the attacks grew shorter, and instead of the condition starting in June with "rose cold," it did not develop until the last of August or the middle of September. It was at this time that the asthma and the nervous phenomena appeared. At first the asthma was severe, but lately it has lessened in intensity, and some years is almost absent.

The nervous phenomena develop at the end of the attack of vasomotor rhinitis and at first they consisted simply of marked excitement. Each year the nervous manifestations increased in severity. The attack almost always develops after a thunder storm in September. In the early spring and summer thunder does not affect her. The duration of the nervous attacks is from two weeks to two months. The patient becomes most excitable, and is afraid that people are trying to poison her and she is fearful of death. Dunbar (Footnote 1, In Osler-McCrae, Vol. II, Page 865) speaks of "a period of depression, often accompanied by suicidal ideas." In this case it seems to be the distinct opposite. She fears that her family are endeavoring to take her property away from her. She imagines she is possessed of great wealth; and frequently when she first goes out after these attacks, she will spend money most lavishly and foolishly. She is extremely talkative, and writes notes to people whom she does not know. As far as is known, she never threatens anyone in these notes, but simply tells them of her troubles and imagined misfortunes. She becomes suspicious of food prepared for her and often refuses to eat. During these attacks she takes aversion to several members of her family, one in particular. When she is well, this particular member is her most constant companion. Her language at times is most obscene and abusive to this sister, and she has tried to strike her at times with various articles. She has a delusion that she is pregnant and is about to give birth to twins. This delusion recurs every year.

Gradually she becomes quieter, sleeps better, and almost in twenty-four hours will be her normal self. These attacks almost always terminate suddenly, but it is some weeks before she regains her normal physical condition. During the winter months the patient attends, without the least difficulty, to her daily duties, which are most trying and exacting, and her general health is excellent.

Physical examination—The patient is a rather thin, poorly developed adult white female.

Eyes—Ocular movements full; pupils equal and react to light and accommodation.

Neck—Normal; no enlargement of cervical or thyroid glands.

Chest—Long and narrow of the phthisical type. Expansion is fairly good and equal. Vocal resonance slightly increased over the right apex anteriorly, with slight impairment in the percussion note, and slight prolongation of expiration. There are a few asthmatic rales scattered over the chest.

The heart is normal in size, action is somewhat rapid, but no irregularities. There are no murmurs.

Abdomen—Negative. The tip of the coccyx is abnormally long and extremely sensitive.

Reflexes—All increased.

The patient has never permitted a thorough examination of her nose and throat, but as far as it has been possible to do so they are negative.

Urine examination negative.

There is practically nothing in the literature on this subject that I have been able to find.

Whether the mental condition following these attacks is the result of the vasomotor rhinitis, or whether it is an independent condition, and their occurrence together is simply coincidence, I do not know. It seems more probable that with its periodic occurrence and that it always follows the attack of vasomotor rhinitis, that there might be some toxic substance developed that acts upon a hypersensitive nervous system and induces these attacks.

302 South Nineteenth Street.

EPITHELIOMA OF THE ESOPHAGUS; CASE REPORT.*

DR. EMIL MAYER, New York.

In the early part of August, 1917, Mr. X., aged 55, a cloakmaker, consulted me for dysphagia which began ten months previous and had become worse until at the time of presentation, when his sufferings were intense.

At first he could swallow all kinds of food, but of late he had to restrict himself to liquids.

*Reported before the New York Academy of Medicine, Section on Laryngology and Rhinology, Nov. 28, 1917.

He had been seen by several laryngologists, none of whom made a diagnosis to him or to any of his family. One thought it was a cancer.

He had been X-rayed, and was told that nothing abnormal was found.

He had lost some in weight, and was feeling weaker than before, but his chief complaint was that of pain associated with inability to swallow solids.

On laryngological examination, as also of the deep pharynx, no evidence of diseased condition presented.

Auscultation of the stomach with the swallowing of liquid showed some retardation from the normal time.

A few days later, under local anesthesia, I introduced a short esophagoscopic tube. A mass high up in the esophagus was seen and a small portion removed for microscopic examination through the tube.

The acting Chief of the Pathological Laboratory at Mount Sinai Hospital, Dr. E. Bernstein, reported that it was a typical epithelial carcinoma.

The mass was large, and almost completely occluded the entrance to the esophagus.

The case was considered inoperable, and the members of his family were advised of the nature of the trouble and gastrostomy suggested as a palliative measure.

After conferring with the patient, who was informed that it was a growth that ought not be operated upon and the suggestion of a permanent stomach opening laid before him, he declined to submit to that procedure.

He was then placed under direct treatment of radium, in the hope that at least the pain might cease, but in this instance no amelioration whatever has followed its use.

Orthoform gives temporary relief.

The special points of interest in this case are as follows:

First: The importance of direct inspection in addition to the other methods of establishing a diagnosis.

Second: Failure of the X-ray to show so large a growth although performed by most competent men.

Third: The non-introduction of esophageal bougies which might readily produce an immediate fatality.

Fourth: Entire absence of relief from radium.

40 East 41st Street.

SOME UNUSUAL DISEASE CONDITIONS APPARENTLY CURED BY TONSILLECTOMY; REMARKS ON POST-OPERATIVE TREATMENT: PRELIMINARY REPORT.

DR. VIRGINIUS DABNEY, Washington, D. C.

That the faucial tonsils are frequently the source of serious systemic infection and may be the very fountain head of metastases is now so well known that I shall proceed on the assumption that this is a conceded fact. Yet, in many of my cases, so innocent was the appearance of the tonsils, and so few were the subjective symptoms elicited, that I would have declined to remove them had not a competent internist already eliminated all other likely sources of infection. It is most important that this step should be first taken in all obscure cases of arthritis, Bright's disease, retinitis and other states where a distant focus might be supplying the toxin causative of the disorder under investigation. There are, of course, perfectly frank cases, where the tonsils are obviously of the toxic type and need no confirmation of their guilt in causing the disease. A laryngologist may feel confident that a given appearance in a tonsil indicates its *possible* responsibility for a disease condition, but unless an internist of experience and judgment has first eliminated other possible causes, he would be showing poor judgment, if not actual reprehensible carelessness, in removing the tonsils. Shambaugh, whose wide experience and ripe judgment commend his opinion to our ready acceptance, says that "the throat specialist ought not to assume the responsibility of passing on these questions. They are problems that fall more properly in the work of the internist and often constitute one of the most difficult problems for him to solve."

Apical tooth abscesses have been shown capable of causing all the metastatic affections formerly attributed to diseased tonsils, and frequently both conditions are found in the same case. Where this coincidence of toxicity occurs, it is my belief that a careful history will reveal the fact in most instances that the teeth were the original source of infection, and, in fact, caused the tonsillar degeneration.

In view of the great sorrow to the patient, and the enormous economic loss to society of blindness, or even marked loss of vision, perhaps the most brilliant result of tonsil removal is that secured in clearing up the effusion in choroiditis and chorioretinitis of infectious origin. These cases so often proceed to blindness or serious

loss of vision, and with such rapidity, that any treatment which arrests the process, and even clears it up, cannot be overestimated. Thus, in some forty-odd cases which were referred to Drs. A. Y. P. Garnett and W. C. Moore for physical examination by various ophthalmologists, the tonsils were found responsible in 35 per cent (teeth 60 per cent, sinuses 5 per cent). It is well to invite attention to the fact that these cases were first seen by the oculist, then the internist and finally the dentist and laryngologist. Treatment was given as indicated by the findings of this group of examiners, with the eye specialist in control. So slight was the manifestation of tonsillar disease in many of these cases that I was reluctant to operate till I had had a consultation with the internist; yet every case showed definite disease areas beneath the plica, after dissection.

Chorioretinitis. The following three cases illustrate three different ways in which a diseased tonsil may create an inflammatory effusion in the eye:

Case 1. S. T. R., male, 24 years old, good general health. Dr. R. S. Lamb reported a mild but definite chorioretinitis with exudate, and suggested an examination of the tonsils and teeth. Teeth negative. Tonsils were removed in part five years before under local anesthesia, but I found a large piece in one fossa, and a small segment completely invested in the plica on the other side. Under ether both remnants were removed with much difficulty, and some pus and caseous material found beneath both capsules. Absorption of the toxin had been inevitable, as the tonsil tissue was absolutely hidden by the scar and mucous surfaces. Cure was complete in six weeks. This case illustrates the futility and even harm of an incomplete operation, aside from any ethical considerations.

Case 2. H. L. W., female, 27 years old, good general health, rather nervous. Eye report by Dr. J. W. Burke: Seven years before had disseminated chorioetinitis in the right eye, but no trouble ever found in the left, though she had had "a black spot before left eye;" exudative chorioretinitis found with great deal of exudate; no syphilis (Wassermann two years before, and no history of it, as well). After thorough examination by internist, I found her tonsils very suspicious and removed them. The next day the exudate was enormously increased which was, of course, entirely confirmatory of the part the tonsils had been playing. In a month the eye was normal. This patient had been examined by a man of international reputation, who handed her over to a rhinologist, who also did eye work; hence it is all the more astonishing that this specialist did not see the tonsillar disease (pus was secured by probing), but even did

a submucous resection on her septum. While the disease in the tonsil was not very obvious, yet the diagnosis by exclusion was so compelling (and a deviation of the septum never yet caused metastatic infection in the absence of sinus disease) that the tonsils should have been scrutinized with the greatest care. Had they been so examined their responsibility would have been manifest. This case illustrates the necessity for judgment and power of observation in an examination.

Case 3. G. D., female, 31 years old, general health good; nervous at times. Eye report by Dr. W. H. Wilmer: Headache for many years after reading, or exposure to glare, unable to read for more than 10 minutes without suffering afterwards well into the next day. At one time did no close eye work for a year. Central chorioretinitis. Physical examination negative; tonsils found diseased only on being drawn out of fossae and probed. Wassermann weakly positive. Removal of tonsils showed extensive follicular disease, and resulted in the eyes being restored to perfect functional health in three weeks. She had been refracted many times in efforts to relieve the pain and weakness of the eyes, but never had had her tonsils or teeth called into question. This case shows merely ignorance on the part of the original oculist. The eyes in all these cases received appropriate local treatment, such as dionin, pilocarpin, iodid of potash, etc., which assisted in hastening absorption, but did not remove the cause of the effusion.

Chronic indicanuria. H. H. H., male, 37 years old, fair general health; more or less tonsil trouble since childhood, but nothing serious. Typhoid fever eight years before, serious case. Indicancria for ten years without interval; constant efforts at eradication without success. For past year and half subnormal temperature, ranging from 95° F. morning, to 97° F. evening. During this period anemic, dispirited and listless. Diagnosis of adrenalin insufficiency made (and very likely true) by competent internist; pulse 60, systolic pressure 95. Consulted me for relief of fullness in ears and slight deafness, subsequently found to be due to congestion of the mouth of the Eustachian tube, an extension of chronic inflammation of the tonsils. These glands were seen to be buried, moderately enlarged and extending very high into the supratonsillar fossa. Catheterization always relieved all symptoms, but only for a few days at a time. Removal of the tonsils, a perfectly obvious necessity, revealed deep disease and considerable pus, and resulted in permanent relief of the symptoms for which the operation was done, as well as of all those previously thought due to the adrenalin

trouble. The chronic presence of indican was not known to me, but, on my remarking on the astounding change in his whole attitude to his work and the marked physical improvement, he mentioned that the test made that day (two weeks after operation) showed the absence of indican from his urine for the first time in ten years; and it has continued absent after some ten months. The urinalysis before operation showed it in great amount.

Multiple Arthritis and Bright's Disease (hyaline casts, albumin, high blood pressure and headache). *Case 1.* M. W. T., female, 43 years old, fair general health; practically all joints involved, pain intense and swelling great; albumin and hyaline casts for ten years; severe migraine for five years; during past two years rarely absent for more than three or four days at a time. Eyes showed chronic disseminated chorioretinitis. Typhoid fever twenty years ago. X-ray of teeth showed numerous suspicious teeth, which had been well taken care of. Tonsils deeply buried, but had never given any serious trouble. However, at operation they were found sufficiently toxic to account for many, if not all of her symptoms. The teeth were extracted but showed no apical abscesses, though much caries in the upper portion. However, all teeth had been regularly filled and treated for years by competent dentists, so I believe they exercised little if any effect on the general condition of the patient. For the first six months after operation the patient had not one of the objective or subjective symptoms, and has had none of them to return save a few headaches in the past two years.

Case 2. M. B. H., female, 45 years old, general health poor, very nervous. Recurrent attacks of mild sore throat and severe arthritis in all large and many small joints, with great swelling and temperature up to 101° F. Teeth defective but well cared for. Hyaline casts, albumin in urine for several years (present day of operation); deposits in retina from old disseminated chorioretinitis; headaches often. Tonsils flat, but showing numerous crypts and some pus on probing. At operation one tonsil showed much detritus, and sharp attack of arthritis was caused in 24 hours. Culture of tonsils gave pure streptococcus, from which a vaccine was made and given for some ten weeks. Convalescence slow and pain left very gradually, but all symptoms are still absent after year and a half. The casts and albumin disappeared in two days and have not returned. Headache is still very rare, though stiffness without swelling or pain in joints occasionally manifests itself.

Darier-Roussy Sarcoid. This term is the only one under which the following case can be classified, in which Dr. H. H. Hazen as-

sured me the lesions are absolutely identical. He referred the case after an examination of the patient from top to toe, including the tonsils, which he said offered the only possible clue to an etiology.

F. C., male, 28 years old, complained of painful nodules upon his arms and legs, situated near large vessels and adjacent to bone, varying in diameter from half to one inch, deeply situated in the corium. Sections showed a pan-phlebitis and pan-arteritis with a surrounding infiltrate of epitheloid tissue (report of Dr. Hazen). In the seven years of his trouble the patient had consulted the best internists and dermatologists in the country, but had never had any relief or any real diagnosis till Dr. Hazen took hold of his case. Repeated Wassermann tests were consistently negative, and I found his tonsils certainly toxic, though not enlarged. Neither Dr. Hazen nor I had any very roseate prognosis for the result of the tonsil removal, but as they were diseased and had given him some slight discomfort in the past, and as his suffering from the nodules was constant and severe, the operation was a conservative and wholly justifiable measure, and such it proved. As usual, the tonsils were found much more diseased than they outwardly suggested, having many minute pockets of pus throughout their substance. In two months all the nodules disappeared and did not return till eight months later, when a gingivitis and pyorrhea alveolaris caused a slight recurrence. This relapse is now under control, and but strengthens the theory of the tonsillar origin, one of the focal variety. The return is thus obviously due to the development of a new focus, and this too is being proved by the disappearance under treatment of the nodules, directed at the gum and alveolar process disease.

Since the almost universal adoption of total removal of the tonsils as the preferred tonsil operation, there has accumulated much evidence that the cosmetic and functional results have been often disappointing and even disastrous in their local effect. In a few cases this is unavoidable, but often the cause lies in failure to treat the operative wound persistently till healing has taken place. Where this neglect has occurred the fossae are invaded with quick growing granulations and their subsequent organization into hard, irritating masses causes discomfort and interference with the vocal function of the tonsillar pillars. The deforming contractures which are seen at times are due to a totally different cause: failure to conserve the pillars at operation, removing their inner surface. After experimenting somewhat I have adopted a definite routine of post-operative treatment of the tonsillar fossae which has given me encour-

agement despite the occasional faulty results. After leaving the hospital the patient reports daily at the office and there the fossae are first sprayed freely with peroxid, the froth is then sprayed away with any bland alkaline wash and, lastly, the now thoroughly cleansed cavities are swabbed with any form of iodin and glycerin one may prefer. For the first two applications a spray is to be preferred to a swab as it penetrates much better, removes the detritus more thoroughly and is much less, if at all, painful. The pain which is always present, and the edema which is nearly always present in some degree, can both be much relieved and at times abolished by hourly normal salt solution irrigation of the fauces at a temperature of about 110° F., beginning three hours after operation and kept up as long as the patient is in the hospital. This treatment possesses the further advantage of materially hastening the healing process and removing the thick tenacious mucus, whose forcible expulsion on the part of the patient is so painful, and whose retention in the mouth so provocative of gagging, itself distressing and apt to lead to hemorrhage. Nasal irrigation is likewise helpful, in that it removes the clots and re-establishes nasal respiration which relieves the discomfort of mouth breathing, one of the lesser causes of the patient's suffering. It is an emergency measure (not routine) and should not be done more than twice after operation, however. The application, while the patient is still in the hospital, of various iodin solutions does not appeal to me, as the fibrinous covering which nature at once throws over the denuded surfaces is distinctly protective and even germicidal, and for this reason should not be disturbed for two or three days, at the end of which time it will have lost these properties and become a slough. This, of course, is no longer protective but an obstruction to the escape of noxious material. My objections to the early application is not only theoretical, as set forth, but eminently practical, as I have tried it faithfully and found it in no wise superior to the technique I have devised myself, one doubtless many others follow without my knowledge. Moreover, the pain it causes is always intense, and sometimes excruciating, which condition causes the reflection that we pay too little attention to the patient's feelings in many of our manipulations. When a patient says a certain procedure hurts, the remark is often not heeded, unless we reply that it is unavoidable; whereas it is generally unnecessary. Grasping the tongue in laryngoscopy, separating the alae of the nose or even depressing the tongue, that simplest of procedures, are all painful or free of pain in proportion to the dexterity or consideration of the examiner. Rough-

ness of manipulation bears no relation to thoroughness, nor does gentleness prevent a searching inspection. Inasmuch as a patient will submit to a more thorough examination and treatment at the hands of an attendant who evinces some desire to avoid unnecessary pain, I submit that this consideration is not purely academic but eminently practical. All patients are not aware that the Stoics regarded pain as one of the lesser pleasures, and probably would not subscribe to the doctrine if they did.

In all cases reported here the frequency with which tonsils outwardly little diseased have been shown as harboring serious toxicity, the source of metastatic disease, is striking. However, I wish to state unequivocally that I am reporting seeming cures and not recommending tonsillectomy for the relief of any of the conditions discussed herein, not excepting arthritis, unless the patient has already been thoroughly examined by a competent internist who has excluded other sources of infection; even so, the tonsils must appear at least abnormal, if not actually toxic. The abuse of the tonsil operation has grown to such an alarming extent since the promulgation of the focal infection theory that I do not wish to appear to lend my support to any increase in the list of diseases which justify tonsil removal. It is my creed that no child's tonsils should be removed unless it is possible to demonstrate a definite connection between those tonsils and some pathologic condition in that child. On the other hand, given an adult suffering from a disorder commonly associated with tonsillar disease, it is justifiable to remove these tonsils if there is a reasonable suspicion attached to them. I have seen four patients die of endocarditis (of proved tonsillar origin) from neglect of this precaution, though none of them had anything more severe when I saw them than a history of repeated attacks of tonsillitis, associated with slight pains in the joints. The tonsils were plainly diseased, though not much enlarged, except in one case. The deaths occurred, one in four years after my advice, the other three within two years, and none of them had any heart lesion or functional irregularity when seen by me. The heart in each instance was examined by a diagnostician of unquestionable ability; so I know the lesions which resulted in death were not present when I suggested operation, and hence I feel sure that the deaths were avoidable. One of the cases was especially sad, in that she was quite willing to submit to operation, but her family physician dissuaded her. It is hardly to be questioned but that her death lies at his door.

1633 Connecticut Avenue.

A CASE OF PANSINUSITIS ON ONE SIDE WITH TIC DOULOUREUX ON THE OTHER; NO OPERATION: NECROPSY: UNUSUAL FINDINGS.

DR. WILLIAM LEDLIE CULBERT and DR. JAMES G. DWYER,
New York City.

On October 6, 1916, I was asked to see Mrs. J. N., at the Neurological Institute in the service of Dr. Joseph Collins. She was a widow, 66 years old, of Swedish descent, family history negative. The patient was a large woman, weighing 240 pounds, had always been very active, and gave no history of illness excepting severe colds each year, and that she had polypi removed from the nose ten years ago.

The present illness dated from an onset of pain in the left side of her face last February. It became very severe and prostrated the patient, and the various doctors whom she consulted gave her little or no relief. The trouble continued with intermissions. In August she took a long automobile ride to the Adirondacks. On this occasion she acquired a severe cold, and from then on the pain in her face became much worse, necessitating her going to bed. Then she was brought down to Albany to the home of her married daughter, where she consulted Dr. Elting, and several other physicians. During a period of about six weeks she received various treatments there, including vaccine, after which she returned home to Brooklyn. Her physician in Brooklyn then gave her two injections of alcohol, one over the left eye, and one in front of the left ear. These only made the pain more severe, and she finally came to the Neurological Institute on October 2nd, where I saw her a few days later.

Her appearance was most dejected, she was groaning with pain, breathing through her mouth, and constantly holding her head; when her hand was removed from her head, she held the left eye shut. She had lost about 40 pounds in weight, and her flesh was soft and flabby. She described the pain as "a tearing pain" in the back of the left eye, and all over the left side of the head. She also complained of a numb area underneath the left eye, which at times became swollen and very cold. On examination I found the head very tender to the touch all over the left side, especially above and below the left eye and in front of the left ear. The least pres-

*Read before the New York Academy of Medicine, December 20, 1916.

sure over the antrum, the malar prominence, the frontal bone, and the anterior part of the temporal bone caused the most excruciating pain. Examination of the nose showed large roomy nostrils, the left side being clear but containing some pus; the right side was completely occluded with polypi, and a small amount of pus. The throat was surprisingly normal. The teeth were all gone, excepting the lower incisors and the canines, which did not appear to be in a very healthy condition. The ears were not involved.

There was complete paralysis of the left external rectus muscle, which had developed in September, just after the alcohol injections. The pupils were equal. The fundi of the eyes were reported normal.

Transillumination showed the left frontal sinus clear; the right, dark; the left antrum, fairly clear; the right, dark. The left pupil showed distinctly; the right did not.

This seemed to indicate that all the trouble was on the right side, although the symptoms of pain, tenderness, and the paralysis of the external rectus were on the left side. On account of diplopia, she had learned to keep the left eye shut.

She was referred to Dr. Caldwell for skiagraphs of the sinuses, which confirmed these findings, and showed that there was a pansinusitis of the right side, but revealed nothing on the left side or in the left orbit.

Dr. Collins considered that she was suffering from two conditions: the pyogenic infection of the right sinuses, and arterial sclerosis with tic douloureux on the left side; and, furthermore, that nothing could be done for the latter condition until the pus infection on the right side was relieved. He therefore requested me to do a right-sided radical Killian operation, and for this purpose she was removed to the Manhattan Eye, Ear and Throat Hospital on October 10. After further deliberation, however, and noting the patient's weak and enfeebled condition, the question arose whether we were justified in operating on such a patient with a decided arterio-sclerosis, a blood pressure of over 200, and a right-sided pansinusitis, with any hope of relieving the symptomatic tic douloureux on the left side. It was decided to postpone operation and to keep her under observation, and in the meantime to pursue further investigations.

A blood examination showed 90 per cent hemoglobin, 4,800,000 red blood cells, 10,000 leucocytes; no abnormalities in the red blood cells; the differential count showed 21 per cent small mononuclear lymphocytes, 72 per cent polynuclear neutrophiles, 5 per cent large

mononuclear leucocytes, and 2 per cent mast cells. In differential blood counts taken for a number of days the relative percentage remained about the same, but the leucocytes ran up as high as 14,000.

The temperature did not run above 99.5; the pulse varied from 68 to 88; the respiration remained about normal; the blood pressure, from 150 to 210.

The urine, while usually normal, at times showed decided traces of albumin. The amount passed in twenty-four hours varied from 20 ounces plus to 45 ounces plus.

Blood and spinal fluid Wassermanns were negative.

Dr. Hutton, the attending physician, was asked to see her. He found her chest and abdominal cavities practically negative. Her reflexes also revealed nothing. Dr. Hutton suggested having her blood examined for urea, uric acid, creatin, creatinin and carbon monoxide. This was done by Dr. Dwyer, without finding any marked abnormalities except a slight increase of the blood content of creatinin. The pheno-phthalein examination test previously done at the Neurological, was also made and revealed only slight variation from the normal.

Aspiration of the right nostril yielded a fair amount of pus; this, she thought, made her head feel somewhat easier.

While these various tests did not diverge far from the normal, she nevertheless presented the appearance of a person so profoundly ill that I was reluctant to do a major operation. On October 14 I asked Dr. George Brewer, consulting surgeon of the hospital, to see her. He was of the same opinion.

On October 18, under local anesthesia, a number of large polypi were removed from the right nostril, and later aspiration was again done. She left the hospital on October 22, going home to Brooklyn. I kept in touch with the case, and when the patient died, just four weeks later, on Sunday, November 19, asked and obtained permission for a cranial necropsy.

I was fortunate in locating Dr. James G. Dwyer, and together we opened the cranium and removed the brain. Dr. Dwyer's report is as follows:

"The examination was limited to the skull. When the calvarium was removed, the dura was found to be thin and adherent to the bone. There were no evidence of meningitis, except in so far as the adhesions of the dura signified an old slow process, probably secondary to pressure. The brain itself had quite a normal appear-

ance, save for the profound atheroma of the blood vessels, especially at the base of the brain.

"The ethmoid sinuses on the right side were quite full of inspissated pus resembling cottage cheese. The right frontal sinus was full of liquid, creamy pus. The sphenoids were continuous and full of inspissated pus enclosed in a membranous sac which did not open into the nose. The left frontal sinus was clear. The left ethmoid sinuses were quite full of pus and there was an opening through the outer wall into the left orbit, and in the left orbit was found a collection of thick pus which bathed the nerves and vessels. There was no exopthalmous of either eye. There were openings from the left ethmoid directly into the left antrum and the latter was full of pus. Its posterior wall was broken down, and this allowed the pus to bathe Meckel's ganglion.

"Cultures of the pus showed pure staphyloccus pyogenes sureus."

16 East 54th St.
40 East 41st St.

LARYNGECTOMY FOR EPITHELIOMA.

DR. H. ARROWSMITH, Brooklyn, New York City.

Mrs. A. S., a widow, was admitted to the service of Dr. Purdy H. Sturges at the Methodist Episcopal Hospital in Brooklyn, January 14, 1917, complaining of dysphagia and spasmodic cough. The cough was worse at night or when lying down. It may be of interest to state that the patient dated the beginning of her symptoms to 1892, twenty-five years ago, and stated that the difficulty in swallowing and cough had been gradually increasing in severity since then. She had not, however, previously consulted a physician.

Physical examination on admission revealed a cauliflower growth, in size about one and one-half centimeters in its greatest diameter, involving and projecting from the right arytenoid posteriorly, accounting for the dysphagia, and also extending across the median line toward the left arytenoid. Both vocal bands were apparently normal. There was no involvement of the ventricles. There was no hoarseness. There was no involvement of the cervical glands.

It was decided to remove a portion of the growth for microscopical examination and diagnosis and this was done at once under cocain anesthesia and suspension. The pathological report showed the growth to be a squamous-celled epithelioma.

Dr. Sturges consulted with Dr. Robert Abbe in regard to the advisability of treatment by the use of radium. As the result of a combined examination by Dr. Abbe and Dr. Voislawsky at St. Luke's Hospital, Dr. Abbe suggested the removal of the growth as thoroughly as possible down to normal tissue and then, after an interval of ten days, the use of radium.

On February 24, two weeks after the removal of this section, the patient was readmitted to the hospital and on the following day, by invitation, under colonic oil-ether anesthesia, I suspended the patient and removed as much as possible of the growth. A preliminary tracheotomy was performed by Dr. Sturges to facilitate the later use of radium and as a safeguard in case of edema following the operation.

At the time of this operation, which followed the removal of the specimen for microscopical diagnosis after an interval of only two weeks, the growth was seen to have more than doubled in size and in view of this fact it was decided by Dr. Sturges and myself that the patient's chances for permanent cure demanded a complete removal of the larynx and plans were accordingly made to do it.

Ten days later, again under colonic oil-ether anesthesia, a laryngectomy, after the method of Keene, was performed by Dr. Sturges. The Keene technique was somewhat modified and slightly complicated by reason of the previous tracheotomy wound. The growth was found to have infiltrated the upper anterior portion of the esophagus in relation with the cricoid cartilage and about one and one-half centimeters of this was removed. A nasal tube for purposes of feeding was introduced previous to suturing the opening in the esophagus. The performance of the operation was greatly facilitated by the use of colonic oil-ether anesthesia and the operation was completed in fifty-five minutes, the patient leaving the table in very good condition.

The post-operative recovery was uneventful except for a hypostatic congestion of the lungs on the eighth day, which lasted for three days. These three days were the only ones during which she was annoyed in any way by mucous accumulation or tracheal secretion.

No difficulty was encountered in properly nourishing the patient. She was given liquid nourishment through the nasal tube for ten days, when it was permanently removed. Following the removal of the tube there was some discomfort in swallowing, which disappeared in two or three days. She was allowed up and out of bed on February 23, eleven days after the operation.

170 Clinton Street.

EMBRYONIC CARCINOMA OF ETHMOID AND ROOF OF ANTRUM.

Dr. J. A. MacKenty, New York City.

U. M., aged 17 years, presented herself for examination on July 6, 1917.

Past History.—Acute coryza with cough and temperature in January, 1917; nasal discharge (yellow from both sides) accompanied by swelling of both eyelids, equal, not tender nor painful. This acute attack lasted nine days. From this time up to the present she has had post-nasal yellow and pinkish discharge. Four weeks prior to her visit to me she noticed a swelling of the right side of face over the antrum and eyelid, and numbness over the distribution of the second division of the fifth nerve for two weeks. Second upper right bicuspid tooth painful and tender.

Examination.—Right external nasal wall congested and the turbinated bodies swollen. Tenderness and swelling over the root of second right bicuspid tooth. Tenderness over external wall of antrum.

Transillumination gives a shadow over the right maxillary and ethmoidal sinuses.

Antrum washing gave inspissated pus.

Diagnosis.—Antrum infection from diseased tooth though the numbness in the distribution of the second division of the fifth nerve could scarcely be explained unless there was bone involvement of the antral roof.

First operation.—Diseased tooth removed and antrum opened inter-nasally. For ten days antrum was irrigated with a return of blood-stained fluid. Pain and tenderness not relieved.

Then I began to suspect a more serious condition, grounding my fears upon the sanguineous character of the antral washings and upon the blocking of function in the second division of the fifth nerve. The possibility of malignancy was explained to the parents and a radical operation advised. There was a suspicion of right eye exophthalmos.

Second operation.—Radical external sinus operation. Antrum was opened through the Killian incision after removing the ethmoid cells. A mass of soft, friable tissue was removed from the antrum

*Read before the New York Academy of Medicine, Section on Laryngology and Rhinology, Nov. 28, 1917.

—its attachment was to the roof. Exophthalmos of right eye now became apparent.

Microscopic examination.—(Made by Dr. James G. Dwyer). This tissue presents the picture of carcinoma made up of cells of a very young type. It may be embryonical in nature. The mass is made up in a very large part of cells, which appear here and there to be rapidly invading the surrounding tissue. I would judge that this growth would recur rapidly and that the recurrence would be of a very malignant type, as once cells of these growths are stimulated, growth is very rapid.

Third operation.—Ten days later the anterior wall of the antrum, external wall of the nasal cavity, and the floor and remaining inner wall of the orbit were removed. This was preceded by terminal ligation of the branches of the external carotid. In ten days between the second and third operations the growth had apparently entirely reproduced itself.

A maximum dose of radium was applied for two hours by Dr. Janeway through the open wound. Then the wound was closed.

Following this operation the improvement was marked. Pain, tenderness, swelling and exophthalmos disappeared. The cavity became clean and apparently was healing. Two months later, October 15, 1917, examination showed a rapid resumption of growth along the external wall of orbit and behind the orbit; internal strabismus and diminished vision. Radium was again applied with marked improvement up to Nov. 1.

The interesting points in this care are: (1) Its resemblence to an ordinary antrum disease from tooth infection.

(2) The suspicious symptom of fifth nerve involvement.

(3) The subsequent suspicious symptoms of sanguineous antral discharge and slight exophthalmos.

(4) The rapidity of the return of the growth after the second operation (ten days).

(5) The retarding effect of radium upon the later recurrence.

A noteworthy point in the history is the acute sinusitis in January followed by a chronic condition during the winter and spring. This irritation undoubtedly lies in a causative relation to the malignancy, perhaps stimulating into activity some dormant embyronic remain.

May 8, 1918—Patient not expected to live more than few weeks. The return is exctensive and rapidly growing.

43 West 54th Street.

ADENOIDS IN ADULTS.*

DR. FREDERICK A. LEWIS, Auburn, N. Y.

The nasopharyngeal vault of the adult presents a cavity fairly cuboidal in form with firm, smooth walls of mucous membrane. On each side the trumpet-shaped extremity of the Eustachian tube projects well into the chamber.

The direction of these tubes being forward, an angular space is left between them and the posterior wall, known as Rosenmüller's fossa. One object of this arrangement of the tubes with their free and expanded ends is undoubtedly to prevent the ingress and retention of mucus.

If this space should be filled in partially or wholly one can readily see that mucus could find its way more easily into these tubes. Also this obstructing body would cause more secretions to be formed from irritation by pressure. Furthermore, if this body were a soft and irregular mass it would greatly help to retain the mucus in this location.

The drainage from the nose is normally backward, passing through this fossa and between the Eustachian tubes. When the fossa is of natural depth and the tube ends free, there is little danger of the secretions gaining entrance even though there might be a considerable excess of them.

Adenoid tissue grows from the vault and posterior surface of the nasopharynx, often extending beyond the level of the tubes. It may fill the entire space of Rosenmüller's fossa, and even hang over or crowd the tubes from above. Thus, what should be the free ends of these tubes may be actually buried in this mass. The tissue is soft and irregular, having crypts and sulci similar to the faucial tonsils.

Keeping the general anatomy in mind, we see that when adenoids are present, the mucus from the nose instead of passing behind and around these tube ends, is thrown directly against and into them.

The adenoid, from its honeycombed formation, makes an excellent abiding place for these secretions, and being lymphoid in character may easily absorb and pass them into the system.

As age advances and there is a tendency for this tissue to shrink, adhesive bands are formed stretching between the posterior lip of the tube and the central mass of the adenoid. This simply changes the form of the irritation, from one of pressure to one of pulling.

*Read before the Medical Association of Central New York, Oct. 18, 1917.

We have been led to believe that complete absorption of this tissue is the rule in adult life, but a careful study of statistics and facts does not bear this out. I believe that in a large proportion of cases, probably over 30 per cent, children who have adenoids not removed, retain enough of this tissue in adult life to cause more or less trouble.

What are some of the resulting symptoms? The crowding or drawing on the Eustachian tubes, together with the increased facility for the entrance of germ-laden mucus, produces a chronic inflammation which gradually extends to the middle ear, and causes otitis media with deafness, tinnitus, etc., so common in advanced years.

There may be direct absorption from the adenoid tissue into the lymphatic system, and any of the numerous diseases commonly ascribed to focal infection might come in this manner.

Direct irritation to the larynx may result from the excessive secretion, or the trouble, such as cough or hoarseness, may be reflex in nature.

Patients frequently complain of catarrh, a constant desire to clear the throat, and a feeling of fullness and obstruction in the nose, often with impairment of vocal resonance.

These growths in adults are quite easy to overlook, as they are often flattened and have a fairly smooth surface.

The amplitude of Rosenmüller's fossa is a good guide when using the throat mirror. With spacious nares one can sometimes make a diagnosis by direct inspection; if not, a probe passed to the posterior pharyngeal wall will indicate as to whether this tissue is firm or boggy.

One case will illustrate some of these points: Mr. A complained of nasal obstruction, catarrh, and throat irritation, with some deafness and ear noises. He had a pronounced deviation of the septum, hypertrophied tonsils and a rather small appearing mass of adenoids. A submucous resection was done on the septum, which gave much improvement in nasal breathing. A little later the tonsils were removed, which resulted in still further clearing up the symptoms; but he still complained of head fullness, with catarrh and ear trouble. The adenoids were then removed, and proved to be much larger than they had appeared *in situ*. The patient claimed that this last procedure had given him more relief than either of the former two.

In an article published by Dr. D. M. Barstow in the *New York Medical Journal*, 1905, he gives the results of 57 cases of adenoids in adults, with the following conclusions:

"Adenoid growths in the adult are much more common than is generally supposed.

"The condition is a frequent cause of nasopharyngeal catarrh. It is also a frequent cause of nasal obstruction, and is the causative lesion in some cases of apparent hypertrophic rhinitis. The cases tabulated show that a thorough examination of the nasophaynx should be made in all cases of ear disease and pulmonary tuberculosis.

"The treatment is not difficult, and the results are occasionally brilliant and rarely unsatisfactory."

Dr. Alice G. Bryant in 1908 read a paper before the Section on Laryngology and Otology, of the American Medical Association, entitled: "Streptococcic Infection of the Pharyngeal Adenoid Tissue in Adults." She reported ten cases in which this infection had its inception in the adenoid tissue. These cases were all of the influenza type and cleared up promptly with the use of postnasal douches.

In the *Revue de Laryngologie*, published in Paris in 1899, S. Jankelevitch has quite an extensive article on this subject. He states "that although less frequent in adults, yet they often provoke disturbances similar to those in the child. In the main, however, the symptoms are somewhat different. Nasal and ear disturbances are most frequent in the child. In the adult reflex phenomena occupy a prominent place." He notes, "persistent cough, expectoration, throat irritation and alterations of taste and smell; also headache and disorders of sleep."

O. Orendorff, in an article published in the *Journal of the American Medical Association* in 1909, gives these symptoms: "High arched palate with adenoid fringe, fauces and pharynx covered with sticky mucus, enlargement of lingual papillae, and nodules on posterior pharyngeal wall." He adds, "There is always more tissue than appears on examination."

Capt. L. I. LeWald, Medical Corps, U. S. A., published an article on Adenoid and Tonsillar Hypertrophy in Recruits, in the *Journal of Military Surgery* in 1910. He says that in the recruits examined the occurrence runs as follows:

18 years of age	40 per cent
20 years of age	36 per cent
22 years of age	25 per cent
24 years of age	22 per cent
25 to 30 years of age	14 per cent
30 to 35 years of age	8 per cent

"Examination with the mirror is the only satisfactory way of

making a diagnosis." He also states "that pathological changes in the ear varying from a diminished light reflex, or slight retraction of one or both membrana tympani, to a well marked chronic catarrhal otitis media, were present in 67 per cent of the cases with adenoids. This striking connection between adenoids and middle ear disease is one that cannot be taken too seriously." Continuing, he says: "It is true that the author's observations show that adenoid tissue does undergo atrophy, but this process is so slow that it does not become an appreciable factor until after the age of 25 years. Cases do extend well into the thirties and even occasionally after 40 years of age. It seems best therefore to disregard entirely the possibility of atrophy in the treatment of adenoids both in the child and in the adult."

Chavasse before the Academy of Medicine in Paris, 1908, reported 3000 men examined and 342 with adenoid vegetations. The removal always had a decided effect on the concomitant affections of the ear and nose. Heart disturbances were noted in 17 of these cases.

Dr. H. I. Davenport, of this city, has made a microscopical examination of several specimens to determine whether any special changes took place in this tissue with advancing age. I sent him the adenoids from four nearly consecutive cases at the ages of 6, 11, 22 and 50 years. The result seems surprising, as Dr. Davenport states that there was no essental difference in these cases; in fact none that could be shown with the microscope. One would naturally look for at least a preponderance of connective tissue. This fact may explain why these lymph structures continue to produce such annoying and pronounced symptoms. It is undoubtedly true, however, that the consistency averages somewhat firmer in advanced life.

In my own experience the removal of this tissue has always given very gratifying results. In several cases where both tonsils and turbinates have been hypertrophied, as well as the adenoids, attention to the latter has produced the more decided improvement.

The operation can usually be performed with a local anesthetic.

I have found the La Force adenoidtome the best instrument, as it is both quicker and cleaner in its execution.

To sum up, we find that adenoids in adults play an important part in affections of the ear, nose and throat, and often of more remote organs.

They may be easily overlooked, and are usually larger than they appear.

It is very unwise to depend on atrophy for their removal.

16 William Street.

EDITORIAL DEPARTMENT

THE DEAF

Their Education—Improvement of Conditions—Responsibilities and Participation of the Profession.

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NEW YORK CITY.

UTILIZING FOR EDUCATIONAL PURPOSES SLIGHT REMNANTS OF HEARING IN VERY DEAF CHILDREN.

For some years past, in this department of the LARYNGOSCOPE and in other publications, we have endeavored to impress upon the medical profession the power its members possess of educating their communities to the possibility and the desirability of improving the educational advantages they provided for their deaf children for the purpose of enabling them to learn to speak and to understand when spoken to without the use of finger spelling or the sign language.

Possessing a broad knowledge of the problem as a result of thirty years of labor in this field, we selected the thing that would do more than any other single reform to improve the lip reading and speech teaching conditions in our public schools for the deaf, namely, the segregation of the orally taught from the manually taught. We devoted our efforts principally to impressing upon our readers the inherent futility of the mixed system and the practical necessity of absolute segregation if satisfactory results are to be obtained from the expenditure of time, effort and money on the part of the states in the teaching of deaf children to speak and understand speech.

This campaign of education has been so successful that the question of improving speech teaching conditions by segregating the orally taught from those with whom finger spelling and the sign language are used is now discussed in every state of the Union, and the actual process of segregation is being prepared for in a number of states where it had never existed before. The matter has now

been taken up by so many people, and is being pushed by others so vigorously, that it seems permissible for us to begin the agitation of another reform, second only in importance to that of segregation in a speech environment of the orally taught deaf pupils.

We want now to plead for greater attention to the training of a considerable number of the pupils in every school for the deaf to use, *for the comprehension of language*, the powers of sound perception which they possess. It is my belief, based upon many years of observation, that approximately one-third of the pupils in every public school for the deaf possess sufficient power of perceiving sounds that lie within the range of the speaking voice to enable them to *learn* to comprehend language through the ear, which is the normal way. These pupils are not now receiving the necessary training to enable them to use this remnant of hearing.

It may seem strange to those who have not had occasion to give the matter special consideration, that a child who can hear the sounds of the speaking voice should require any special attention to enable him to learn to understand what he hears. The reason lies in the fact that the child is too deaf to hear speech at ordinary conversational distances, and so does not hear the spoken words when the ideas they express are in his mind, which was the way you and I learned to understand the English language. But the intensity with which the sound affects the hearing mechanism varies inversely as the square of the distance between that mechanism and the source of the sound. Translated into figures, this means that a child who cannot hear a word spoken a yard from his ear, may be able to hear that word if it is spoken an inch from his ear, since his ear will then be affected 1296 times as powerfully as it was when the word was spoken a yard away. And if you speak twice as loudly an inch from his ear as you spoke a yard off you will make 2592 times as much impression on his hearing organs.

A child who is so deaf as to require words to be *shouted* an inch from his ear will never spontaneously learn to understand language or to speak, *but he can be taught to do both.* There have always been many such children in the schools for the deaf, and they grow up with little or no speech and understanding of speech, or less than they might have acquired if full advantage had been taken of their remaining power of sound perception.

Not long ago I was in the beginner's classroom in a state school for the deaf. I had been there for an hour and, at the request of

the teacher, had attempted to start two or three of the babies on the road to hearing spoken language. When the teacher had resumed her regular oral work the superintendent came in, and in the course of conversation I spoke of my intention of trying to awaken those in charge of the schools to an increased interest in the slight hearing powers possessed by many of their pupils. He looked around at the nine little ones in the room, who had been in school about three months, and said, "All of these are congenitally totally deaf." I asked to be allowed, when the attention of the children was fixed upon the teacher, to stand behind the class and clap my hands. Instantly, one of the little girls shrugged her shoulders nearly up to her ears and turned around with a questioning look upon her face. The superintendent said, "That was probably a coincidence. I do not think she heard it." A few moments later I tried it again with the same result, and that time he decided that she heard it. As a matter of fact, a short time before he came in I had, in ten minutes, succeeded in getting that little child to distinguish with considerable certainty between three words spoken repeatedly an inch from her ear while the objects for which the words stand were before her. It would take time, patience and some intelligence to build up a hearing vocabulary in that little girl's brain, but it could be done.

There is apt to be some confusion in the minds of people unfamiliar with this matter with regard to just what takes place. This is a purely educational operation. It is not a process of increasing the power of hearing, but of training the brain to interpret sounds into ideas. In all probability the actual power of sound perception will not have increased in the case of a child with whom this process, continued through a school year, has succeeded in creating a hearing vocabulary of many words and sentences. It is only that the child's brain has been educated to associate ideas with the sounds that he was just as capable of hearing at the beginning as at the end of the year. If the reader of this editorial were suddenly transported to a country of whose language he was ignorant he would understand nothing of what was said to him. Not because he could not hear the words, but because his brain had never been educated to associate those sounds with ideas. If he hears the words and sentences often enough, *when the ideas they stand for are in his mind,* he will learn to understand. It has not, however, been a process of learning to hear, but of learning to associate ideas with sounds he could hear just as well at the beginning as at the end.

These little deaf children, who yet have some power of sound perception, never get a chance, under ordinary conditions, to develop this association of ideas with sounds, because they do not hear well enough to get the sounds at the distances they are uttered in ordinary intercourse. Therefore, as they get nothing of value through their ears they soon come to ignore even the sounds they can perceive, and their actual physical deafness is increased by what we might call a psychological deafness. The same psychological deafness that we ourselves manifest when we sit reading or otherwise occupied in a room with a chiming clock, and yet are not conscious that the clock has struck.

I do not need to dwell upon the great superiority of this natural means of access to the understanding of the child through his ears when it can be used. This is the line of least resistance. The line of inherited tendencies. The line of ancestral brain development. When it is not open to us we must use the untrodden and unfamiliar road of the eye in comprehending spoken language, but the ear is the quickest and most practicable route when it is open, or can be opened. I believe it can be opened in the case of about one-third of the pupils in our schools for the deaf; if the children that compose that third can have ten or fifteen minutes a day of the right kind of training.

In order that these children may get this training it is necessary to make the superintendents and teachers believe it can be done and that it is desirable to do it. An intelligent physician in the vicinity of the school can exert a great influence in bringing this about, and I bespeak the co-operation of everyone in a campaign to secure the utmost use of the small amounts of hearing possessed by many deaf children, which at present are neglected and unused.

1 Mt. Morris Park, West.

CHANGE OF NAME OF THE "IODINE POWDER," DESCRIBED IN THE LARYNGOSCOPE, NOVEMBER, 1917.

Since my article appeared in the above issue, my attention has been called by the originator, Dr. N. Sulzberger, of New York, that a preparation with a similar name is on the market. In order to avoid confusion, Dr. Sulzberger has discontinued the original designation for his product, and has requested myself and others to do likewise. He wishes from now on that his powder be known as *"Iodine Powder," (Sulzberger).* Sincerely, M. D. Lederman, M. D.